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HUNGER IN THE INFANT*

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Cannon and Washburn,[1] and Carlson and his colaborers have given us a proved method for studying hunger objectively; its time of occurrence, its intensity, its effects, and the means by which it may be produced or inhibited. They have shown that contractions of the so-called empty stomach cause the hunger sensation. These contractions depend in part on vagus tonus. They can be increased by chemical changes in the blood, but are primarily due to a gastric mechanism as purely automatic as is that of the heart.

Impulses set up by these contractions and carried to the higher centers are, in the normal consciousness, recognized as hunger. These impulses produce secondary effects such as restlessness and irritability. They increase the reflex excitability of the central nervous system, the heart beats faster, and there are changes in the vasomotor mechanism. Well fed, sedentary adults seldom experience hunger. The prime factor in their desire for food depends not on the basis of distress due to the contractions of a hollow viscus, but on "the memory processes of past experience with palatable foods." This psychic factor is appetite, and its absolute distinction from the physical factor, hunger, must be kept in mind.

Working on dogs, Patterson, in 1914, showed the gastric hunger contractions to be much more frequent and vigorous in young than in older animals. In 1915, Carlson and Ginsburg described the great intensity of hunger contractions in the human new-born. Previous to that year no productive analytic studies of the hunger sense in the human infant had been made. Appetite and hunger were not distinguished, and the sucking mechanism alone had been analyzed.

In 1888, Auerbach distinguished the infantile type of sucking from the voluntary inspiratory type employed by the adult, and in 1894 Basch, disproving the older theory of Preyer that sucking is instinctive, showed it to be entirely reflex.

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* Submitted for publication Aug. 18, 1917.
* From the Department of Pediatrics, University of Minnesota.
1. All references to the literature will be found at the end of the article.

Czerny, in 1893, observed that an infant awakened a short time after taking his fill from the maternal breast, would again suck vigorously if placed on it, and concluded that sucking per se could not be considered as a sign of hunger. A few years later (1900) Keller wrote that, since the normal infant sleeps three hours after nursing, although its stomach is empty in two hours, the emptying of the stomach cannot be considered a positive criterion of need for food. Pies, in 1910, considered the reddening and eczema of the lower lip which occurs in undernourished infants as a sign of hunger, and referred it directly to the infant's fruitless sucking. In 1913 Schlossmann concluded from extensive observations on semistarved infants that the sensation of hunger exists only in the imagination.

Meyer and Rosenstern studied the results of starvation in the different types of alimentary disorder, recording particularly the pulse, temperature, respiration and weight changes. Rosenstern later (1911-1912) wrote extensively on the general subjects of hunger and inanition in infancy. These studies are all defective in that they do not distinguish the various factors concerned. Neumann, Pfaundler, Cramer, Süszwein, Barth, and Kasahara have discussed the subject of disturbances in the food urge largely from the point of view of imperfections in the sucking mechanism.

The present studies are concerned particularly with the gastric factors in the urge for food. The major of these, the hunger contractions, was studied by means of apparatus similar to that used by Carlson. A rubber balloon of about 20 c.c. capacity attached to one end of a small soft rubber catheter is inserted into the stomach and inflated, the catheter is attached to a bromoform manometer with a cork float and a writing pennant which records the gastric movements on smoked paper.

The material investigated included 5 premature infants weighing from 1,200 to 2,500 gm., 40 full term new-borns under 3 weeks of age, and 11 older babies, 5 between 1 and 2 months, 2 between 3 and 4 months, 3 between 4 and 6 months, and 1 boy of 2 years with a surgically induced gastric fistula made necessary by the effects of corrosive in the esophagus. The gastric movements of some of the infants were recorded only once; on others as many as twenty observations were made.

Carlson and Ginsburg refer to the readiness with which most infants accept and retain the tube and balloon. It is naturally impossible to secure a graphic record of the stomach movements of a raging infant. Carlson and Ginsburg did their work on full term new-borns. These infants, as a rule, sleep quietly when not disturbed. The present work was carried on in a dimly lighted, quiet room. I had less difficulty when the infant was left undisturbed in his crib than when I

attempted innovations, such as threading a pacifier on the tube or having the infant held in the nurse's arms.

The older babies resent the presence of the tube, and with them it was often necessary to make repeated attempts to secure tracings. Some infants finally became accustomed to the presence of the tube and slept quietly, particularly if the experiments were conducted in the evening. Most of the tracings on the 2-year-old boy with the gastric fistula were made when he was awake. The greatest problem was to keep him sufficiently interested to prevent crying and restlessness and at the same time to prevent riotous hilarity. In his case the balloon was introduced directly through the fistula.

It is said that passage of the stomach tube in infants is apt to cause aspiration pneumonia. No ill results followed the procedure carried out in these studies.

Does the presence of the balloon in the stomach act mechanically to produce gastric contractions? Carlson states definitely that it does not, and gives the following reasons for his belief:

> 1. The presence of the distended balloon in the stomach between the contraction periods does not induce these contractions.
>
> 2. In Mr. V. [his gastric fistula case] the gastric contractions can be observed directly through the large fistula without any balloon in the stomach.
>
> 3. The contraction periods come on just as frequently without any balloon in the stomach and produce the same effect (hunger).
>
> 4. In pigeons the periodic strong contractions of the empty crop can be seen directly through the skin, and a balloon in the crop does not alter their frequency or intensity.

The results of this work fully confirm Carlson and Ginsburg's report that the stomach of the new-born infant exhibits greater hunger contraction than does that of the adult. The intervals between the contraction periods are often less than five minutes and usually not longer than from ten to twenty minutes. The first contraction period after a nursing is apt to consist of from five to twenty separate contractions and to last from two to eight minutes. The succeeding contraction periods frequently endure from thirty minutes to an hour or even longer. The duration of each contraction is about twenty seconds. In many of the infants the contraction time of the more powerful contractions, especially in those periods ending in partial tetanus, was about eighteen seconds. Except in the first contraction period after a nursing, endings in partial tetanus were frequently observed. Partial tetanus is sometimes present before the close of the period. With the apparatus used, the force of the single contractions usually sufficed to raise the column of bromoform 2 to 3 cm. During partial tetanus the bromoform may be raised 5 cm.

Patterson found practically continuous hunger contractions in premature pups. It is particularly easy to obtain graphic records of the

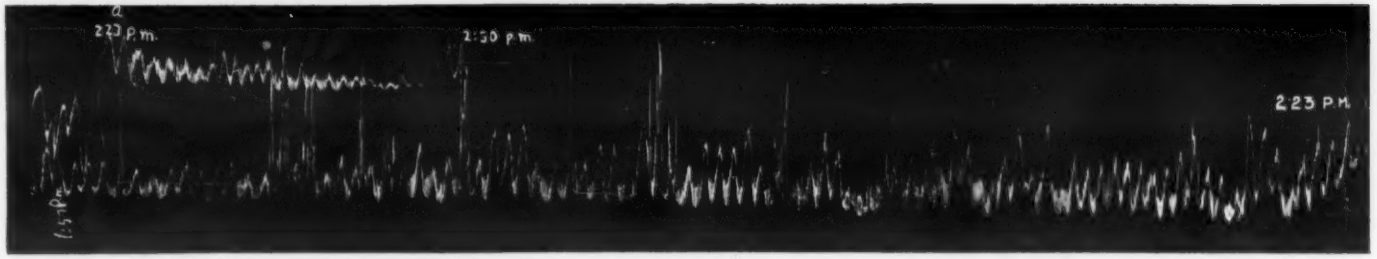

Fig. 1.—Hunger contractions in normal, new-born infant. Beginning of partial tetanus at extreme right in lower tracing.

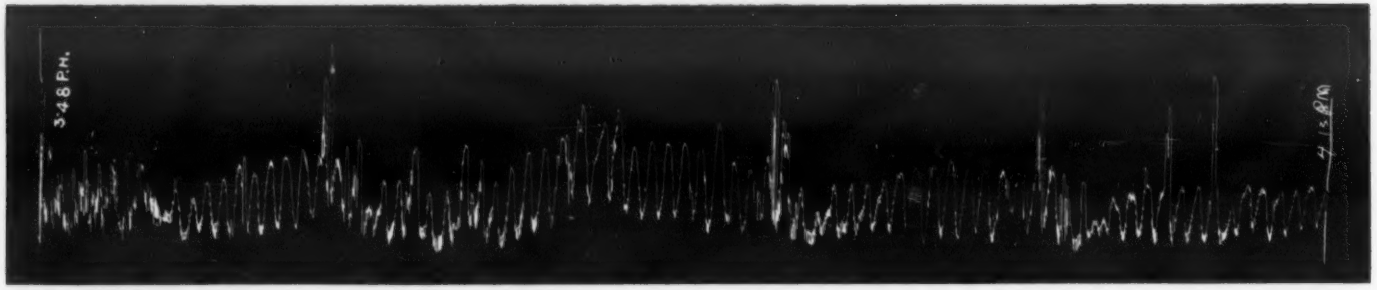

Fig. 2.—Prematurely born infant. Typical activity of the premature baby's stomach. Boy F., aged 15 days; weight, 1,536 gm.; getting 45 c.c. breast milk six times a day. Previous feeding at 2:30 p. m.

hunger contractions of the somnolent, prematurely born infant. The stomach of such an infant is in a state of nearly continuous contraction. The individual contractions require about the same length of time for their completion and are as powerful as those of the full term infant. In a tracing begun forty minutes after a feeding of 15 gm. of breast milk to a premature baby (Baby 5) weighing 1,510 gm., the record appears very like that obtained by Rogers from the crop of a pigeon in the second day of starvation. The periods of contraction last two or three minutes, with intervening periods of quiescence of about the same length. The individual contractions last twelve to fifteen seconds and raise the bromoform column 3 to 4 cm. Partial tetanus is frequent. Nine days later, when the infant was receiving more food, in spite of the fact that he had not gained in weight and that the tracing was begun five hours after his last feeding, the record obtained was similar to those from other infants.

Are the hunger contractions more frequent or more powerful in cyanosed infants? May they furnish a stimulus for crying with consequent better aeration of the lungs? In two such cases no significant increase or decrease in the hunger contractions could be observed. No records were taken from any cyanosed premature infants, although such infants are frequently slightly blue for the first few days.

Carlson, working on the adult, was unable to produce hunger contractions by any sort of stimulus acting directly in the mouth or in the stomach, except that he occasionally could, by suddenly distending the stomach, produce a few transitory contractions. He found, uniformly, that the only effect of such local stimulation was inhibitory. In general, the taste of salt, sour, bitter, or sweet; or the chewing of agreeable, disagreeable, or indifferent substances, all produce temporary inhibition of the gastric contractions. Chewing palatable foods by the adult when hungry causes an inhibition, made more lasting by the flow of appetite juice in the stomach.

Carlson found that acid and alkaline solutions, food and liquids in the stomach, all inhibit the hunger contractions. Inhibition from the stomach is less transitory than that from the mouth. Boldyreff showed that the periodic contractions of the empty stomach were inhibited by the presence of acid in the intestine. Brunemeier and Carlson completed and enlarged this work. They demonstrated inhibition from the presence of gastric juice or acid chyme in the small intestine. This inhibition from the stomach and intestine is reflex, partly through Auerbach's plexus, but mainly through the long reflex arc with the efferent path to the stomach muscles through the splanchnics.

Inhibition from the mouth is not present in the frog (Patterson). Carlson, who suspects that such inhibition involves conscious cerebral processes, has suggested experiments in infants to settle the point.

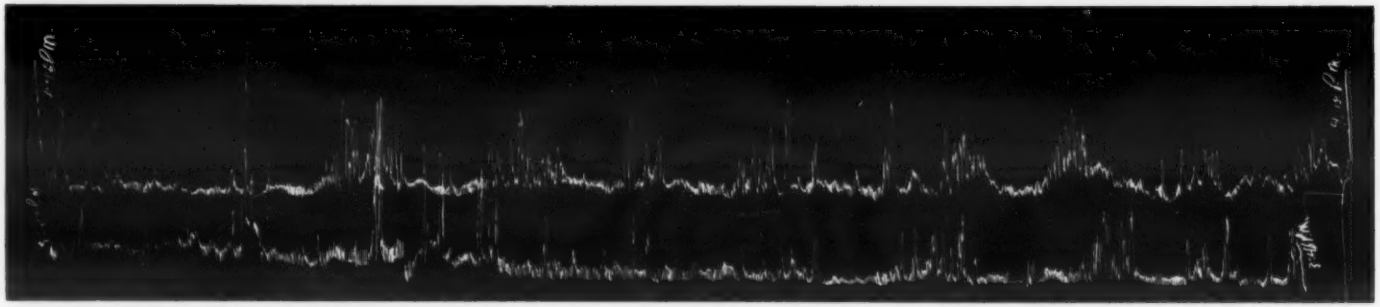

Fig. 3.—Prematurely born infant; shows numerous short, forceful contraction periods. Boy Von, 4 days old; weight, 1,510 gm.; birth weight, 1,715 gm.; at 2:30 p. m. received 15 c.c. breast milk.

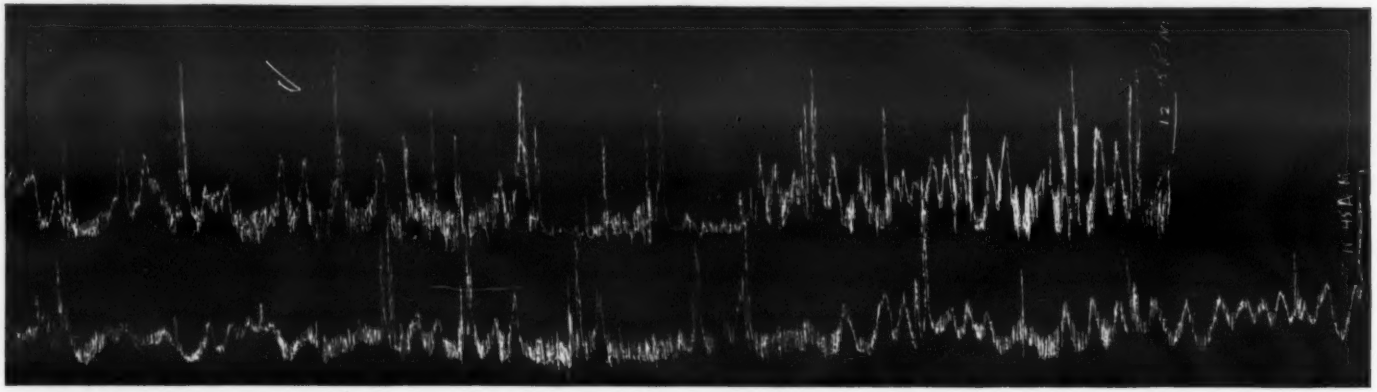

Fig. 4.—Baby Mi., 15 days old; tracing shows presence of hunger contractions in an infant who nursed poorly.

Repeated trials with breast milk, sugar water, common salt, quinin, and lemon juice in the mouths of premature and new-born infants in my study failed to produce inhibition of hunger contractions.

In general I obtained the same results in an infant of 8 weeks. A transitory inhibition occurred occasionally when sugar water was placed in his mouth. In none of the infants did chewing or sucking on the thumb or tube produce inhibition. Nor did such movements or the presence of sugar, breast milk or other substances in the mouth induce hunger contractions.

The boy of 2 years showed inhibition when sugar or protein milk (his diet at the time) was placed in his mouth. Quinin, dilute hydrochloric acid, small amounts of sugar water, table salt in crystals or solution, did not inhibit. Benzosulphinidum solution inhibited twice. It was not used subsequently. The sight of sugar did not inhibit. He began to cry when he saw his bottle if the latter were not given him immediately. Conseqeuntly the effect of his seeing the bottle on the hunger contractions could not be registered. During the periods of quiescence the sight of the nurse who fed him did not induce hunger contractions, although he began to whine and tease when she entered the room.

Apparently inhibition from the mouth was produced by those substances only which the child regarded as food. Quinin very evidently made a profound sensory impression, but did not inhibit the contractions. Dilute hydrochloric acid did not inhibit, while unsweetened protein milk (which is slightly sour) did.

Carlson's hypothesis as to the need of conscious cerebration for the production of inhibitory reflexes from the mouth would appear to offer the correct explanation. It seems to be agreed that the new-born infant leads a subcortical reflex existence (Soltmann-Cramer). Kussmaul and Thiemich note that the new-born infant accepts sugar and rejects salt, food that is sour and bitter — action which is almost certainly purely reflex on the part of the infant.

My work shows that when 20 c.c. of water or milk are introduced into the stomach during a contraction period inhibition follows invariably. This was found true in infants of all ages. With small amounts of water the inhibition often lasted only three or four minutes, when the contraction period would be resumed.

On the other hand, it was not unusual to recover from 15 to 40 cm. of clotted milk through the stomach tube even an hour after vigorous hunger contractions had begun. This is a considerable portion of the infant's meal, and in these cases would represent from one-sixth to one-fourth of his total intake at the previous feeding. Soltmann showed that the inhibiting nervous mechanism of the heart is much less effectual in the new-born infant than in later life. It seems possible

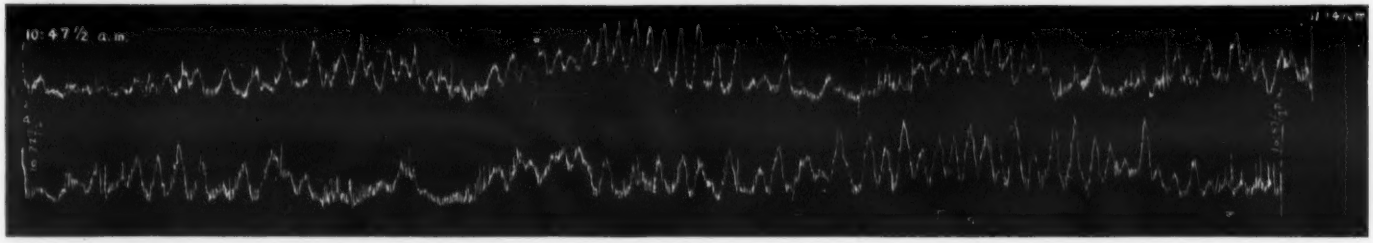

Fig. 5.—Baby Ri., 6 days old, with congenital myxedema; tracing shows presence of hunger contractions in a babe who nursed poorly.

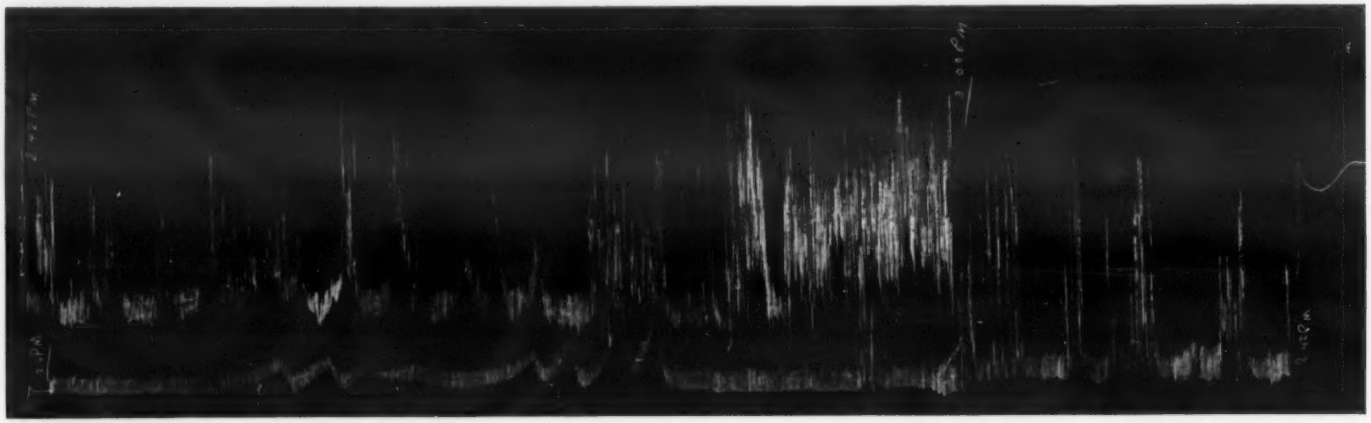

Fig. 6.—Baby J., 4 months old. Tracing shows development of hunger contractions during the interval following a feeding.

that the nervous apparatus for the inhibition of the gastric hunger movements may likewise be immature. Or the tissue hunger may be so great as to overcome any but the strong inhibition of a heavily laden stomach and duodenum.

The vagi form the sensory pathway from the stomach to the brain. The first reflex centers are the sensory nuclei of the vagi in the medulla. A second center, possibly that for conscious hunger, is located in the optic thalami. Rogers has shown that the picking reflex in the pigeon (analogous to the sucking reflex in the babe) is abolished on removal of the thalami.

The reflex irritability (as indicated by the knee jerk) is increased synchronously with each hunger contraction (Carlson). No observations have been made on the infant's knee jerks during the hunger state; but Zybell has shown that the electrical irritability increases during the first eighteen hours of starvation.

Let us summarize the events from the close of one meal till the end of the next. The infant sleeps. The upper stomach musculature maintains a tonic grasp on the contained food. The pyloric antrum is traversed by peristaltic waves (Cannon). The stomach gradually empties. The point of origin of the peristaltic waves rises higher and higher. The tonus rhythm of the fundus begins. The stomach empties itself more completely, the tonus rhythm becomes more intense, and the first hunger contractions appear (Rogers and Hardt).

The first contraction period is apt to be short. After a wait of perhaps twenty minutes a longer and more intense hunger period arrives; then another and another. The infant's sleep becomes lighter. He is more easily awakened by external stimuli or by gastric discomfort. He is put to the breast, nurses vigorously, becomes fatigued (Schmidt, Cramer, Pfaundler), or experiences satiety from distention (Neisser and Bräuning) and again goes to sleep.

What constitutes the hunger state? Does it result from the summation of impulses with an increasing psychic and reflex irritability? The evidence is to the contrary. The increase in the reflex excitability is synchronous with the contraction phase of the stomach, and is absent in the intervals between the contractions. In the infant who has been some hours without food the hunger contractions are nearly continuous, and it would be expected that the reflex excitability would be nearly continuously high.

In the absence of hunger contractions the infant often sucks vigorously on the tube attached to the balloon. The receptive mechanism for the institution of the sucking reflex is so delicate that it is impossible to provide, artificially, a minimal stimulus. During the hunger state, when presumably a rapid succession of hunger contractions maintains a low reflex threshold, there may often be observed a succession of

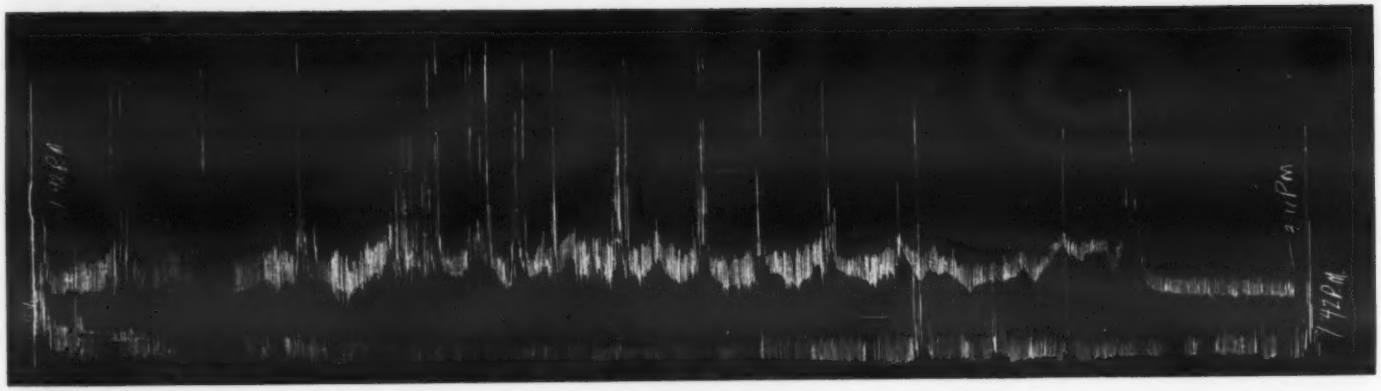

Fig. 7.—Same as Figure 6.

automatic sucking movements involving the lips, tongue and lower jaw, each movement providing the necessary stimulus for its successor.

The lay mind is prone to think that the crying infant is hungry. Comby and Czerny and Keller believe that hunger is a minor cause of crying. Rosenstern notes that in hunger young babies are usually quiet, but that the older infants cry more. Schlossmann remarks that the normal infant endures hunger well.

Observations on this point extending over sixteen months of study of the hunger sensation lead me to believe that in normally thriving breast fed infants, except when more than three or four hours have elapsed since the last feeding, neither the hunger contractions themselves nor the increased irritability due to them are ordinarily immediate factors in the production of crying. Young infants sleep throughout strong contraction periods. Older infants often do the same, and are frequently quiet even from twelve to sixteen hours after a feeding. Mental factors produce crying at a very early age. And the fact that crying ceases when food or water is administered may only mean that the infant's attention is diverted to the performance of a pleasurable act.

It may be noted in this connection that the 2-year-old boy was happier when allowed to take food into his mouth, and that his outlook on life was much more cheerful on days when he could take nourishment by mouth than on other days when the esophageal constriction increased, and it became necessary to introduce the food through the gastric fistula. This feeding through the fistula was without pain, and the child submitted to it with some pleasure.

What is the time interval between feeding and the first appearance of gastric hunger contractions? Ginsburg, Tumpowsky and Carlson studied this point in thirty normal breast fed infants under 4 weeks of age. They gave no data as to gain in weight and did not determine the amount of food taken, but stated that the babes nursed till satisfied. They found the average time between nursing and the appearance of hunger contractions to be two hours and forty minutes, with a minimum of two hours and twenty minutes and a maximum of three hours and thirty minutes. My observations on twelve new-born infants under like conditions yielded these results; a minimum of one hour and thirty minutes, a maximum of three hours and thirty minutes, and an average of about two and one-half hours.

Many infants in the first two weeks do not receive a sufficient supply of breast milk. This is particularly apt to be true of the time the babe and the mother remain in the hospital. Consequently, observations made under the conditions so far outlined may be misleading. Table 1 (A, B and C) gives the results of all satisfactory tracings obtained from normally thriving babies on whom sufficient data as to food intake and weight gain were obtained.

TABLE 1.—Interval for Development of Hunger

A. PREMATURE INFANTS

Name	Age, Days	Food	Feeding Interval	Quantity at Feeding	Interval Before Tracing	Time for Development of First Hunger Period	Remarks
Sw.	7	Breast milk	4 hours 5 times a day	35 c.c.	None	40 min.	Premature Wt. 2,140 gm.
Sw.	11	Breast milk	4 hours 5 times a day	50 c.c.	20 min.	1 hr., 20 min.	Wt. 2,240 gm.
Sw.	20	Breast milk	4 hours 5 times a day	90 c.c.	52 min.	2 hours	Wt. 2,470 gm.
Sw.	25	Breast milk	4 hours 5 times a day	75 gm.	38 min.	1 hr., 50 min.	Wt. 2,565 gm.
St.	13	Breast milk	4 hours 5 times a day	30 c.c.	None	1 hour	Premature Wt. 2,270 gm.
St.	14	Breast milk	4 hours 5 times a day	50 c.c.	None	40 min.	Wt. 2,280 gm.
St.	25	Breast milk	4 hours 5 times a day	90 c.c.	1 hr., 13 min.	1 hr., 15 min.	Wt. 2,450 gm.
St.	28	Breast milk	4 hours 5 times a day	90 c.c.	38 min.	1 hour	Wt.
St.	36	Breast milk	4 hours 5 times a day	100 c.c.	32 min.	1 hr., 50 min.	Wt. 2,720 gm.
Fre.	9	Breast milk	4 hours 6 times a day	45 c.c.	1 hr., 38 min.	1 hr., 38 min.	Premature Wt. 1,380 gm.
Fre.	15	Breast milk	4 hours 6 times a day	45 c.c.	1 hr., 18 min.	1 hr., 18 min.	Wt. 1,530 gm.
Fre	16	Breast milk	4 hours 6 times a day	45 c.c.	23 min.	1 hr., 15 min.	Wt. 1,535 gm.
Fre.	21	Breast milk	4 hours 6 times a day	65 c.c.	2 hours	2 hr., 20 min.	Wt. 1,710 gm.

B. FULL TERM NEW-BORN INFANTS

H.	8	Breast milk	4 hours 5 times a day	?	1 hr., 22 min.	3 hr., 50 min.	Wt. 3,500 gm.; gain in 4 days, 90 gm.
Dav.	8	Breast milk	3 hours 8 times a day	75 gm.	1 hr., 55 min.	2 hr., 25 min.	Wt. 4,160 gm.; gain in 5 days 260 gm.; 10 c.c. of milk clot removed from stomach 1 hr. after beginning of hunger contractions
Dav.	9	Breast milk	4 hours (for preceding 24 hours) 5 times a day	?	2 hr., 30 min.	2 hr., 30 min.	Wt. 4,200 gm.; gain in 6 days, 300 gm.; ½ c.c. thick mucus removed from stomach ½ hr. after beginning of hunger contractions
Wes.	8	Breast milk	4 hours 5 times a day	100 gm.	1 hr., 30 min.	3 hr., 10 min.	14 hours fast previous to last feeding
A.	9	Breast milk + cow's milk } aa 8% lactose }	3 hours 8 times a day	70 gm.	1 hr., 45 min.	2 hr., 10 min.	Wt. 3,610 gm.; gain in 6 days 230 gm.; 20 c.c. of thick soft milk clot removed from stomach 50 min. after beginning of hunger contractions
A.	10	Breast milk + cow's milk } aa 8% lactose }	.....	70 gm.	1 hr., 52 min.	2 hours	15 c.c. of fluid and curds removed from stomach 30 min. after beginning of hunger contractions
Wal.	10	Breast milk	4 hours 5 times a day	95 gm.	1 hr., 30 min.	4 hours	Wt. 3,240 gm.; gain in 6 days, 260 gm.
D.	12	Breast milk	4 hours 5 times a day	115 gm.	2 hr., 30 min.	2 hr., 30 min.	Wt. 3,720 gm.; gain in 7 days, 400 gm.

C. NORMAL INFANTS OVER TWO WEEKS OF AGE

Gor.	18	Breast milk	4 hours 5 times a day	85 gm.	2 hr., 23 min.	4 hours	Wt. 3,500 gm.; gain in 13 days, 70 gm.
Gor.	19	Breast milk	4 hours 5 times a day	110 gm.	1 hr., 58 min.	3 hr., 25 min.	
Way.	Mos. 3.5	Buttermilk + flour + saccharose	4 hours 5 times a day	150 c.c.	2 hr., 27 min.	3 hr., 12 min.	Wt. 4,960 gm.; gain in 7 days, 440 gm.
Way.	3.5	Buttermilk + flour + saccharose	4 hours 5 times a day	150 c.c.	57 min.	3 hr., 20 min.	40 c.c. of thick white materal removed from stomach 20 min. after beginning of contraction period
Way.	3.5	Buttermilk + flour + saccharose	4 hours 5 times a day	150 c.c.	2 hr., 3 min.	4 hr., 35 min.	
Herm.	4	Breast milk	2 or 3 hours irregular	?	2 hr., 45 min.	3 hr., 30 min.	Well nourished; gaining in weight; normal baby; cared for at home; not in hospital
J.	4	Breast milk	5 times a day	?	2 hr., 15 min.	3 hr., 30 min.	Well nourished; gaining in weight; normal baby; cared for at home; not in hospital
Ad.	4	Malt soup	4 hours 5 times a day	125 c.c.	2 hr., 37 min.	3 hr., 30 min.	Wt. 3,500 gm.; gain in 2 weeks, 200 gm.; prematurely born; 1,600 gm. at birth

The time required for the development of hunger in the premature infant is noticeably short. In the case of the full term new-borns the figures obtained agree fairly well with those given by Ginsburg, Tumpowsky and Carlson, but are definitely greater than those obtained by me (mentioned in a preceding paragraph) from infants whose food intake was not accurately known.

The time required for the development of hunger in any one infant is fairly constant over a short period of time, provided the amount and kind of food is not changed. This conclusion rests not only on the results shown in Table 1, but on a dozen other observations on infants whose feeding conditions remained constant during the time in which studies were made.

With the older infants difficulty in maintaining quiet, after the insertion of tube and balloon, limits the number of observations which give positive evidence as to the first appearance of hunger contractions. Many less successful observations on healthy, normally developing infants yield this negative evidence that in such infants more than a month old I did not observe the development of hunger before the end of three hours.

It should be noted further that the contraction period, the first appearance of which is recorded in Table 1, is the first one to develop after feeding. This period is usually short and is not made up of forceful contractions. With Infants J. and A. more intense and more nearly continuous contractions did not begin for four and four and a half hours, respectively.

Habits as to feeding interval affect the time required for the development of hunger chiefly as they influence the emptying time of the stomach. It has been shown that the speed of gastric emptying is proportional to the length of time during which the individual has been without food (Tobler, Haudek and Stigler), and that large feedings are emptied with relatively greater rapidity than small ones (Tobler and Bogen). Habits undoubtedly exert a more powerful influence on the mental factors associated with appetite than on hunger itself.

Tables 2, 3 and 4 illustrate the shorter time required for the development of hunger in infants with chronic nourishment disturbance, and indicate that the presence of hunger contractions is not in itself evidence that the stomach is ready for food.

In the columns headed "Remarks" in Tables 1, 2, 3 and 4, there are notes as to material recovered with the stomach tube after the onset of gastric hunger contractions. In normal babies, however, there probably does exist a relation between the emptying time of the stomach and the interval for the development of hunger.

Observations on the emptying time in infants, so far reported, have been made either with the relatively stiff catheter, the stomach tube,

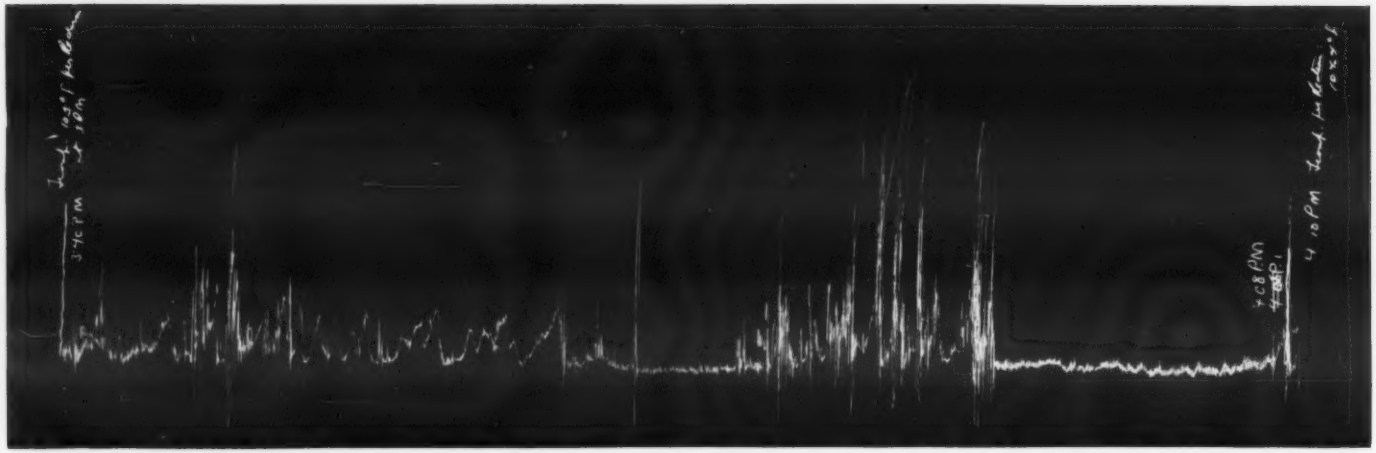

Fig. 8.—N. N., a 2-year-old boy with typhoid fever. Hunger contractions present when rectal temperature ranges between 104.4 and 105 F.

or the Roentgen ray. The flexible tube introduced by Rehfuss should replace the catheter for this purpose; it was used in my work. The literature contains no reports of the time required for gastric digestion in the premature infant. The emptying time in normal breast-fed infants under 1 week is usually less than one hour (Leo). The Roentgen-ray observations of Ladd and of Tobler and Bogen would indicate that in normal breast-fed infants the stomach is frequently not empty until after two to three hours. The figures obtained with the use of the stomach tube by Epstein, Czerny, Keller and Cassel indicate a delayed emptying time in gastro-intestinal disease.

TABLE 2.—HUNGER IN ATROPHY RESULTING FROM CONTINUED STARVATION

N. N., aged 2 years; gastric fistula; weight fluctuating between 6,800 gm. and 7,200 gm.; typhoid fever Dec. 1 until Dec. 14, 1916.

Date 1916	Food	Time of Last Feeding	Beginning of Ttracing	
10/19	Diluted cow's milk + general diet	6 a. m.	10:15 a. m.	Practically continuous hunger periods until 10:50 a. m.
10/20	Diluted cow's milk + general diet	10 a. m.	2:12 p. m.	Practically continuous hunger periods until 3:15 p. m.
11/ 1	.....	10 a. m.	2:16 p. m.	One hunger period at 2:45 p. m. to 2:50 p. m. Hunger contractions practically continuous after 3 p. m. until 4:50 p. m.
11/10	160 c.c. cow's milk at preceding feeding	2 p. m.	2:45 p. m.	Observations continued until 5:10 p. m. No hunger periods, but child cried or was restless over half of the time
10/11	160 c.c. cow's milk at preceding feeding	2 p. m.	9:00 p. m.	Practically continuous contractions until 11 p. m.
11/11	200 c.c. protein milk + 7% dextrimaltose	5 p. m.	10:12 p. m.	Practically continuous contractions until 11 p. m.
11/12	200 c.c. protein milk + 7% dextrimaltose	5 p. m.	8:41 p. m.	First hunger period at 9:30; practically continuous from 9:45 on until 10:44 p. m.
11/29	200 c.c. protein milk + 7% dextrimaltose	1 p. m.	4:02 p. m.	First hunger period began at 4:35 p. m.; practically continuous from 5 p. m. on until 5:42 p. m.
12/27	200 c.c. protein milk + 7% dextrimaltose + cereals	9 a. m.	12:34 p. m.	Hunger periods practically continuous until 1:30 p. m.
12/28	200 c.c. protein milk + 7% dextrimaltose + cereals	9 a. m.	12:22 p. m.	Hunger periods began about 12:50; hunger periods became continuous after 1:20 p. m.
12/30	200 c.c. protein milk + 7% dextrimaltose + cereals	5 a. m.	10:13 a. m.	Practically continuous hunger periods until 11:20 a. m.

Major, using the Roentgen ray, finds the emptying time delayed in dyspepsia, but accelerated in decomposition. With the same method Pisek and LeWald found the emptying time to be shorter in infants with chronic disturbances of nutrition.

These last findings, taken in conjunction with the already quoted reports of Tobler, and of Haudek and Stigler, that the emptying time is shortened by hunger, are suggestive of the results here obtained experimentally; that is, the greater gastric hunger contraction in infants with chronic nourishment disturbance.

Not only is the interval for development of hunger shorter in such infants, but the contractions become much more intense. Nov. 10,

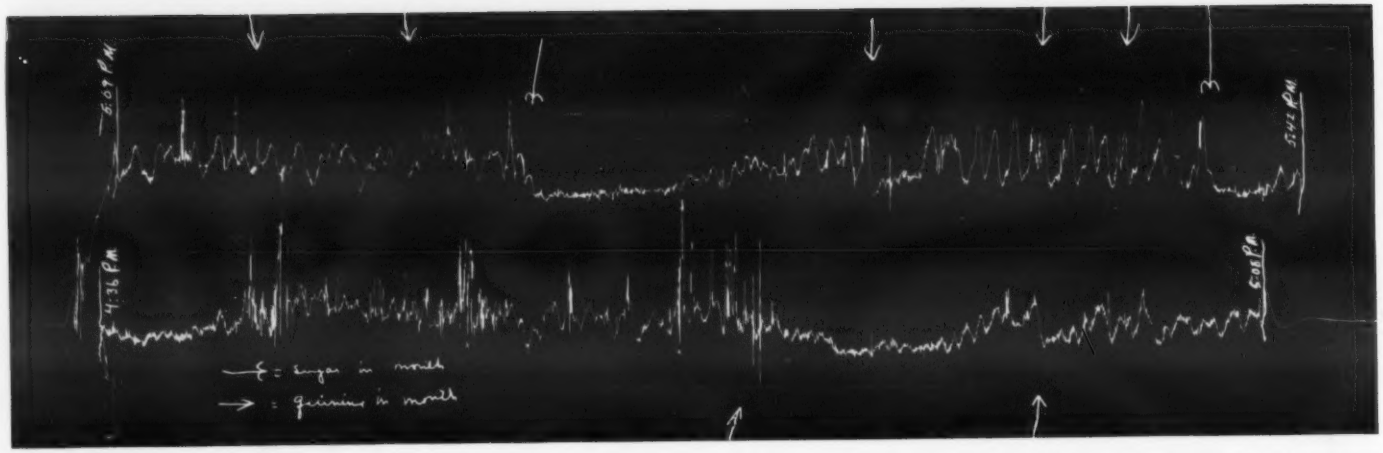

Fig. 9.—N. N., 2 years old. Tracing shows inhibition from sugar in the mouth; absence of inhibition from quinin in the mouth.

1916, the 2-year-old boy (Table 2), whose weight in spite of a calorically sufficient intake had remained stationary, and whose temperature had been irregular, developed fever and diarrhea. After eight hours of starvation, with temperature normal, the graphic record of his gastric activities resembled those of the starving pigeon and of the premature infant already mentioned. The contractions were continuous and required only twelve seconds for their completion. Next day the child was put on protein milk and thereafter improved.

It is generally agreed that mixtures with high fat content leave the stomach most slowly, while those with low fat and high carbohydrate

TABLE 3.—HUNGER IN EXUDATIVE DIATHESIS

Aus., 7 weeks old, Dec. 19, 1916. Feeding interval 4 hours, 5 times a day.

Date	Weight, Gm.	Food: Breast Milk + Buttermilk + Flour + Saccharose, C.c.	Time of Last Feeding	Beginning of Tracing	
12/19/16	4.450	100	1 p. m.	3:28 p. m.	Hunger contractions began at 3:30 p. m. Hunger contractions continuous from 3:55 to 4:37 p. m.
12/22/16	4,460	100	9 a. m.	11:49 a. m.	Hunger contractions present at 11:49 a. m. Hunger contractions continuous until 12:50 p. m.
12/22/16	4,460	150	1:15 p. m.	3:34 p. m.	Hunger contractions began at 5:45 p. m.
1/ 6/17	4,640	125	8 a. m.	10:55 a. m.	Hunger contractions began at 10:55 a. m. Hunger contractions continuous until 12:21 p. m.
1/ 8/17	4,640	125	8:40 a. m.	11:51 a. m.	Hunger contractions began at 12 m. Hunger contractions strong and continuous after 12:18. Babe was restless
1/11/17	4,650	125	8:45 a. m.	11:11 a. m.	Hunger contractions present by 11:45 a. m. Hunger contractions continuous until 1:35 p. m.
1/13/17	4,740	150	8:20 a. m.	11:03 a. m.	Hunger contractions began at 11:45 a. m. Hunger contractions continuous from 12 to 1 p. m.
1/15/17	4,770	150	8:45 a. m.	11:01 a. m.	Hunger contractions present at 11:20 a. m. Hunger contractions continuout from 11:30 a. m. until 12:50 p. m.
1/15/17	4,770	150	12:50 p. m.	3:21 p. m.	Babe cried a large part of time; no evidence of hunger periods until 5 p. m.

leave most rapidly. In Infants A. and W. the time interval for the development of hunger contractions was much longer when they received low fat and high carbohydrate, and shorter when they received high fat and low carbohydrate. This would be paradoxical if the gastric hunger contractions depended exclusively on the emptying time.

It is, then, only in normal babies, receiving well tolerated food in sufficient quantity, that the development of hunger waits on the emptying of the stomach.

The interval necessary for the development of hunger depends in part on the form of nourishment and is shortest with that food which least satisfies the infant's tissue need (Table 4). The question as to whether the rapid development of hunger in qualitatively poorly

Fig. 10.—Same child as in Figure 9. The tracing shows continuous short contractions in atrophy; after eight hours of starvation.

nourished infants depends on the administration of food deficient in carbohydrate in particular, or on the giving of food poorly tolerated in general, is not answered. Records of the gastric contractions in infants suffering from the chronic nourishment disturbance due to long continued carbohydrate overfeeding (the "Mehlnährschaden" of Czerny) would help to settle this point.

TABLE 4.—INTERVAL FOR DEVELOPMENT OF HUNGER IN INFANTS WITH CHRONIC DISTURBANCE OF NUTRITION AND SHOWING INFLUENCE OF CHANGE IN FORM OF NOURISHMENT

Name and Date	Age, Mo.	Diagnosis	Weight, Gm.	Food	Interval Before Tracing	Time for Development of First Hunger Period	Remarks
Til.	3	Atrophy.....	Stationary 4,080	120 gm. protein milk + 7% dextrimaltose 5 times a day	.....	.....	Hunger periods with partial tetanus less than 3 hours after feeding
Ad. 2/26/17	4	Chronic alimentary disorder due to overfeeding with milk	3,300	150 c.c. ½ milk + 10% saccharose 5 times a day	.....	.....	Hunger contractions present in 2 hours and 20 minutes
Ad. 3/ 8/17	4	Chronic alimentary disorder due to overfeeding with milk	3,500	125 c.c. malt soup 5 times a day	.....	.....	Babe improved clinically; hunger contractions first appear in 3½ hrs.
Way.	3	Chronic nourishment disturbance with eczema	.....	Mixture containing 8% fat. Feeding intervals short and irregular. After entrance to hospital fed 5 times a day	.....	.....	Entered hospital April 14, 1917
4/14/17	..	.....	4,200	150 c.c. ½ cow's milk + 10% saccharose	2 hr., 7 min.	2 hr., 7 min.	Stomach emptied 3 hours after feeding; ½ c.c. of mucus and thick curd obtained
4/23/17	..	.....	4,510	150 c.c. ½ cow's milk + 10% saccharose	2 hr., 10 min.	2 hr., 10 min.	Eczema increased; 40 c.c. of clotted milk and clear thin fluid recovered from stomach 3 hours and 25 minutes after feeding
5/ 1/17	..	.....	4,960	150 c.c. buttermilk + flour + saccharose	2 hr., 3 min.	4 hr., 35 min.	Eczema has disappeared
5/ 2/17	..	.....	4,960	150 c.c. buttermilk + flour + saccharose	2 hr., 27 min.	3 hr., 12 min.	
5/ 3/17	..	.....	4,960	150 c.c. buttermilk + flour + saccharose	57 min.	3 hr., 20 min.	40 c.c. of thick white material removed from stomach 20 minutes after beginning of first contraction period

Attention has already been called to the heightened electrical reactions found by Zybell in hungry infants. I also wish to mention the findings of Finklestein, Thiemich and Japha that the electrical irritability is frequently heightened in artificially fed infants, and of Czerny and Moser that there is an increase in the electrical irritability of infants suffering with "Mehlnährschaden." It is possible that the heightened electrical irritability in all depends on the increased hunger contractions due again in part to the constant chemical stimulation reaching the stomach from the semistarved tissues.

Most premature infants and many young infants nurse poorly. The consequent effect on lactation and on the babe's nourishment is serious. An extensive literature on this subject has been developed in German, but there is surprisingly little in French and in English.

In 1888 Auerbach described the infantile manner of sucking, which depends on the chewing muscles, and Escherich showed its teleologic importance. The reflex paths and center in the medulla were demonstrated in 1894 (Basch). Cramer, Suszwein, Finklestein, Rott, Rosenstern, Barth and Kasahara have further studied the question and report results which in general support the theory that the inability to nurse well is to be attributed primarily to an imperfect nervous mechanism and not to muscular weakness.

For further elucidation of the question, tracings of the movements of the empty stomach were taken in two infants who were extreme examples.

The first baby (Baby M.), weighing 2,700 gm. at birth and presenting no anatomic peculiarities, took very little from the mother's breast during the first three weeks, although sufficient milk was expressed therefrom to feed the baby and to complement the feedings of other babies.

The second infant (Baby T.), aged 3 months, had weaned himself from the breast, had developed dyspepsia and atrophy on artificial feeding, and could be made to take his food from the bottle only with great difficulty. He seemed able to fix his attention on anything other than the act of feeding.

In these infants as well as in the five prematures, and in one typical case of congenital myxedema, hunger contractions of at least normal force and duration were present. At the time they were studied, none of the infants was able to nurse successfully. In all, the sucking reflex was qualitatively present.

This study does not solve the problem as to the causation of feeble nursing, but does limit the field of possibilities by excluding derangements of the primitive hunger apparatus.

Carlson reports Rupp's finding that hunger contractions persist during the fever excited by the administration of typhoid vaccine. The boy with the gastric fistula contracted typhoid fever from a carrier. Tracings taken while his rectal temperature ranged between 104.4 F. and 105 F., show the presence of hunger contractions.

Carlson and Ginsburg found hypertonicity and hypermotility in the stomachs of two infants with pylorospasm and stenosis. From a six weeks' old infant (Baby S.) with pyloric stenosis, I obtained records which agree with Carlson and Ginsburg's description of periods of tetanus lasting several minutes interspersed with vigorous contractions of normal duration.

Carlson suggests that pylorospasm and stenosis may be an expression of gastric hypermotility. His cases were seen late, as was the one here reported. In the absence of tracings taken at the beginning of the disease, it is likely that the hypermotility results from the inanition following the obstruction at the pylorus. And without definite knowledge that the stomach was washed empty, the long periods of tetanus observed may represent the so-called visible gastric peristalsis.

SUMMARY

The study of fifty-six infants from birth to 2 years of age gives the following results:

1. Confirmation of previous work, that hunger contractions are greater in the new-born infant, with description of these contractions.

2. Determination of the still greater hunger contraction in the stomachs of prematurely born infants, with description of these contractions.

3. There is no relation between cyanosis and hunger contractions.

4. Inhibition of the hunger contractions from the mouth does not occur in young infants.

5. Inhibition of the hunger contractions from the mouth in older infants is present only as the result of stimuli, which the babe has learned to recognize as food. It does not occur with substances producing equally strong sensory impressions, but which are not considered by the infant as food.

6. Inhibition from the mouth is psychic in character.

7. Reflex inhibition from the presence of food in the stomach is present in infants of all ages.

8. This reflex inhibition from the stomach may be only partially developed in young infants.

9. Successive automatic sucking movements — each sucking act serving as the stimulus for its successor — are present during the hunger state, when the reflex threshold is kept almost constantly low by a rapid succession of hunger contractions.

10. In normally developing breast fed babes, hunger is not ordinarily an immediate cause of crying.

11. The average time required for the development of hunger in healthy infants gaining in weight and receiving a known sufficient amount of food is, in prematures, under one month, one hour and forty minutes, with a maximum of two hours and twenty minutes and a minimum of forty minutes; in full term infants under two weeks, two hours and fifty minutes, with a maximum of four hours and a minimum of two hours; in infants from two weeks to four months,

three hours and forty minutes, with a maximum of four hours and thirty-five minutes and a minimum of three hours and twelve minutes (Table 1).

12. The time required for the development of hunger in any one infant is fairly constant over a short period of time provided the amount and kind of food is not changed (Tables 1, 2, 3 and 4).

13. The time required for the development of hunger in infants with chronic nourishment disturbance is shorter than in normal infants (Tables 2, 3 and 4).

14. The time required for the development of hunger is shorter when the infant receives food which is poorly tolerated (Table 4).

15. Hunger contractions occur in these infants long before the stomach has emptied. Consequently their presence is not in itself an indication that the stomach is ready for food.

16. The feeble nursing exhibited by most prematures and by many older infants is not due to derangement of the primitive hunger apparatus. Hunger contractions are present and of normal intensity in such infants.

17. Hunger contractions were present in one infant with congenital myxedema.

18. Hunger contractions were present in a 2-year-old boy with typhoid fever when the rectal temperature ranged between 104.4 F. and 105 F.

19. Confirmation of previous findings of increased hunger contractions in infants with pyloric stenosis.

I wish to express my sincere thanks to Dr. A. J. Carlson of the University of Chicago for suggestions which aided materially in carrying out these studies; to Dr. E. P. Lyon and Dr. A. D. Hirschfelder for the loan of apparatus from the departments of physiology and pharmacology; to Dr. F. H. Scott and Dr. F. B.. Kingsbury for advice and assistance in the construction of apparatus; to Dr. F. W. Schultz for the use of material from the Infant Welfare Clinic; to Dr. N. O. Pearce, teaching fellow in pediatrics, and to head nurses Barber and Wenck, who cheerfully assisted in preparing the little patients for examination.

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REFERENCES

1. Auerbach, L.: Zur Mechanik des Saugens und der Inspiration. Arch. f. Anat. u. Physiol., 1888, p. 59. Quoted by Uffenheimer: Ergebn. d. inn. Med. u. Kinderh., **1,** 307-309.

2. Barth, H.: Untersuchungen zur Physiologie des Saugens beim normalen und pathologischen Brustkindern. Ztschrc. f. Kinderh., 1914, **10,** 129.

3. Basch, K.: Die Centrale Innervation der Saugbewegungen. Jahrb. f. Kinderh., 1894, **38,** 68.

4. Boldireff, V. N.: Periodic Work of the Digestive Apparatus in an Empty Stomach, Arch. Soc. de biol., 1905, **11,** 1. Quoted by Carlson, Footnote 9.

5. Brunemeier, E. H., and Carlson, A. J.: Contributions to the Physiology of the Stomach; XIX. Reflexes from the Intestinal Mucosa to the Stomach, Am. Jour. Physiol., 1914-1915, **36,** 191.

6. Cannon, W. B.: A Consideration of the Nature of Hunger, Harvey Lecture, Phila., 1911-1912, **7,** 130.

7. Cannon, W. B., and Washburn, A. L.: An Explanation of Hunger, Am. Jour. Physiol., 1911-1912, **29,** 441.

8. Cannon: The Mechanical Factors of Digestion, New York, Longmans, 1916.

9. Carlson, A. J.: Contributions to the Physiology of the Stomach; I. The Character of the Movements of the Empty Stomach in Man, Am. Jour. Physiol., 1912-1913, **31,** 151.

10. Carlson, A. J.: Contributions to the Physiology of the Stomach; II. The Relation Between the Contractions of the Empty Stomach and the Sensation of Hunger, Am. Jour. Physiol., 1912-1913, **31,** 175.

11. Carlson, A. J.: Contributions to the Physiology of the Stomach; III. The Contractions of the Empty Stomach Inhibited Reflexly from the Mouth, Am. Jour. Physiol., 1912-1913, **31,** 212.

12. Carlson, A. J.: Contributions to the Physiology of the Stomach; IV. The Influence of the Contractions of the Empty Stomach in Man on the Vasomotor Center, on the Rate of the Heart Beat, and on the Reflex Excitability of the Spinal Cord, Am. Jour. Physiol., 1912-1913, **31,** 318.

13. Carlson, A. J., Orr, J. S., and McGrath, L. W.: Contributions to the Physiology of the Stomach; IX. The Hunger Contractions of the Stomach Pouch Isolated According to the Method of Pawlow, Am. Jour. Physiol., 1914, **33,** 119.

14. Carlson, A. J., and Ginsburg, H.: Contributions to the Physiology of the Stomach; XXIV. The Tonus and Hunger Contractions of the Stomach of the New-Born, Am. Jour. Physiol., 1915, **38,** 29.

15. Carlson, A. J., and Ginsburg, H.: Contributions to the Physiology of the Stomach; XXX. The Tonus and Contractions of the Empty Stomach of Infants With Congenital Pyloric Stenosis, Pylorospasm and Chronic Vomiting (Merycism), Am. Jour. Physiol., 1915, **39,** 310.

16. Carlson, A. J.: The Control of Hunger in Health and Disease, University of Chicago Press, 1916, p. 228.

17. Carlson, A. J.: The Control of Hunger in Health and Disease. See Footnote 16.

18. Cassel, J.: Zur Kenntnis der Magenverdauung bei Atrophia infantum, Arch. f. Kinderh., 1891, **12,** 175.

19. Comby, J.: Traite des malades de l'enfance, Paris, Masson, 1902, p. 5.

20. Cramer, H.: Ueber die Nährungsaufnahme der Neugeborenen. Deutsch. med. Wchnschr., 1900, **26,** 32.

21. Cramer: Quoted by von Reuss, A.: Die Krankheiten des Neugeborenen, Berlin, 1914, Enzyklop. d. klin. Med., p. 66.

22. Czerny, A.: Die Ernährung des Säuglings auf Grundlage der physiologischen Functionen seines Magens, Prag. med. Wchnschr., 1893, **18,** 495, 510.

23. Czerny, A., and Moser, P.: Klinische Beobachtungen an magendarmkranken Kindern im Säuglingsalter, Jahrb. f. Kinderh., 1894, **38,** 430.

24. Czerny and Keller: Des Kindes Ernährung, 1906, Part 1, p. 582.

25. Epstein, A.: Ueber Magenausspülungen bei Säuglingen, Arch. f. Kinderh., 1883, **4,** 325.

26. Escherich: Ueber die Saugbewegung beim Neugeborenen, Sitzungsb. d. Gesellsch. f. Morphol. u. Physiol. in München, 1888, **4,** 72.

27. Finklestein, H.: Lehrbuch der Säuglingskrankheiten, Berlin, Kornfeld, 1905.

28. Ginsburg, H., Tumpowsky, I., and Carlson, A. J.: The Onset of Hunger in Infants After Feeding; A Contribution to the Physiology of the Stomach, Jour. Am. Med. Assn., 1915, **64,** 1822.

29. Haudek, M., and Stigler, R.: Radiologische Untersuchungen über den Zusammenhang zwischen Austreibungzeit des normalen Magens und Hunger gefühl, Arch. f. d. ges. Physiol., 1910, **133,** 145.

30. Kasahara, M.: The Curved Lines of Suction, Am. Jour. Dis. Child., 1916, **12,** 73.

31. Keller, A.: Ueber Nährungspausen bei der Säuglingsernährung, Centralbl. f. Inn. Med., 1900, **21,** 393.

32. Kussmaul: Untersuchungen über die Seeleben des Neugeborenen, Quoted by von Reuss, 1859, p. 65. Die Krankheiten des Neugeborenen, Berlin, 1914.

33. Ladd, M.: Gastric Motility in Infants as Shown by the Roentgen Ray, Am. Jour. Child. Dis., 1913, **5,** 345. Ibid., The Influence of Variations of Diet on Gastric Motility in Infants, Arch. Pediat., 1913, **30,** 740.

34. Leo, H.: Ueber die Function des normalen und kranken Magens und die therapeutischen Erfolge der Magenausapülung im Säuglingsalter, Berlin. klin. Wchnschr., 1888, **25,** 981.

35. Luckhardt, A. B., and Carlson, A. J.: Contributions to the Physiology of the Stomach; XVII. On the Chemical Control of the Gastric Hunger Mechanism, Am. Jour. Physiol., 1914-1915, **36,** 37.

36. Major, R.: Roentgenologische Beobachtungen am Säuglingsmagen, Ztschr. f. Kinderh., 1913, **8,** 340.

37. Meyer, L. F., and Rosenstern, J.: Die Wirkung des Hungers im den verschiedenen Stadien der Ernährungsstörung, Jahr. f. Kinderh., 1909, **69,** 167.

38. Neisser, E., and Bräuning, H.: Ueber normale und über verzeitige Sättigung, München. med. Wchnschr., 1911, **58,** 1955.

39. Neumann, H.: Ueber erschwerte Nährungsaufnahme bei kleinen Kindern, Therap. Monatsh., 1893, **7,** 220. Abstr. in Arch. f. Kinderh., 1897, **22,** 152.

40. Patterson, T. L.: Contributions to the Physiology of the Stomach; XIII. The Variations in the Hunger Contractions of the Empty Stomach With Age, Am. Jour. Physiol., **33,** 423.

41. Patterson, T. L.: Contributions to the Physiology of the Stomach; XXXVI. The Physiology of the Gastric Hunger Contractions in the Amphibia and the Reptilia, Am. Jour. Physiol., 1917, **42,** 56.

42. Pfaundler: Ueber Saugen und Verdauen, Wien. klin. Wchnschr., 1899, **12,** 1012.

43. Pies: Zur Physiologie des Neugeborenen; Ueber die Dauer, die Grösse und den Verlauf der physiologischen Abnahme, Monatschr. f. Kinderh., 1910, **9,** 514.

44. Pisek, G. R., and Le Wald, L. T.: The Further Study of the Anatomy and Physiology of the Infant Stomach Based on Serial Roentgenograms, Am. Jour. Dis. Child., 1913, **6,** 232.

45. Preyer: Die Seele des Kindes. Quoted by Baach, K.: Jahrb. f. Kinderh., 1894, **38,** 68.

46. Quest, R.: Ueber den Einfluss der Ernährung auf die Erregbarkeit des Nervensystems im Säuglingsalter, Wien. klin. Wchnschr., 1906, **19,** 830. Quotes: Finklestein: Fortschr. d. med., 1902, **20**; Thiemich: Revue d' hyg. et de méd. d. inf., 1903, **2**; Japha, A.: Ueber den Stimmritzankrampf der Kinder. Berl. klin. Wchnschr., 1903, **40,** 1126.

47. Rehfuss, M. E.: A New Method of Gastric Testing, With a Description of a Method for the Fractional Testing of the Gastric Juice, Am. Jour. Med. Sc., 1914, **147,** 848.

48. Rehfuss, M. E., Bergheim, O., and Hawk, P. B.: Gastro-Intestinal Studies; The Question of the Residuum Found in the Empty Stomach, Jour. Am. Med. Assn., 1914, **63,** 11.

49. Rogers, F. T., and Hardt, L. L. J.: The Relation Between the Digestion Contractions of the Filled, and the Hunger Contractions of the "Empty" Stomach, Am. Jour. Physiol., 1915, **38,** 274.

50. Rogers: The Hunger Mechanism in Birds (Preliminary Report), Proc. Soc. Exper. Biol. and Med., 1916, **13,** 119.

51. Rosenstern, I.: Ueber Inanition im Säuglingsalter, Ergebn. d. inn. Med. u. Kinderh., 1911, **7,** 332.

52. Rosenstern, I.: Hunger im Säuglingsalter und Ernährungstechnik, Deutsch. med. Wchnschr., 1912, **38,** 1834.

53. Rott: Zur Ernährungstechnik frügeborener Säuglings, Ztschr. f. Kinderh., 1912, **5,** 134.

54. Schlossmann: Ueber das Verhalten des Säuglings im Hunger. Verhandl. d. Versamml. d. Gesellsch. f. Kinderh. . . . deutsch. Naturf. u. Aerzte, 1913, Wiesb., 1914, **30,** 143.

55. Schmidt, A.: Brustsaugen und Flaschen saugen. München. med. Wchnschr., 1904, **51,** 2141.

56. Soltmann: Experimentelle Studien über die Funktionen des Grosshirns der Neugeborenen. Jahrb. f. Kinderh., 1876, **9,** 106.

57. Soltmann: Uber das Hemmungsnervensystem des Neugeborenen. Jahrb. f. Kinderh., 1877, **11,** 101.

58. Süsswein, J.: Zur Physiologie des Trinkens beim Säugling. Arch. f. Kinderh., 1904, **40,** 68.

59. Thiemich, M.: Ueber die Diagnose der Imbecillität im frühen Kindesalter. Deutsch. med. Wchnschr., 1900, **26,** 34.

60. Tobler: Ueber die Verdauung der Milch im Magen. Ergebn. d. inn. Med. u. Kinderh., 1908, **1,** 495.

61. Tobler and Bogen, H.: Ueber die Dauer der Magenwerdauung der Milch und ihre Beeinflussung durch verschiedene Faktoren. Monatschr. f. Kinderh., 1908-1909, **7,** 12.

62. Zybell: Beiträge zur Behandlung der Spasmophilie. München. med. Wchnschr., 1911, **58,** 2357.

HUNGER AND APPETITE SECRETION OF GASTRIC JUICE IN INFANTS' STOMACHS*

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There is apparently a gastric element in appetite. The contractions of the stomach institute hunger. Its profuse and rich secretion causes an entirely different sensation — not painful, but pleasant. Carlson concludes that the appetite or psychic gastric juice described by Pawlow[1] stimulates sensory nerve endings in the gastric mucosa. The resulting sensation resembles that which follows the first few mouthfuls of good food at a meal to which one has come hungry, and directs the flow of consciousness toward the matter of taking food.

Pediatric literature contains many references to this secretion. Bauer and Deutsch found no gastric juice in the baby's stomach after it had reached eagerly for its bottle. Pfaundler noted that in babes who nursed actively the stomach emptied sooner, and the degree of acidity attained was higher than in babes who were fed passively or through the tube. Cohnheim and Soetbeer, working with gastrotomized new-born pups, obtained juice containing hydrochloric acid even when the pups nursed on nonlactating breasts. A. H. Meyer found a great variation in gastric acidities in the same child and conjectured that the variations might depend on the presence or absence of Pawlow's appetite juice. Schmidt writes that the infant on the breast works and stimulates the secretion of gastric juice. Meisl advocates the use of a pacifier before meals to cause the flow of appetite juice. Bogen, whose material included a 3½-year-old boy with a stenosed esophagus and gastric fistula, concludes that psychic secretion of gastric juice does occur. Nothmann, in 1909, formally investigated the question of the secretion of appetite juice by the infant's stomach, and concluded that it took place even immediately after birth. Rosenstern advised the use of pepsin and hydrochloric acid to stimulate the appetite of infants who nurse poorly. Bönniger could find in pups no relation between the kind of food and the secretion of gastric juice.

With the exceptions of the work done on pups, and Bogen's work on a 3½-year-old boy, the foregoing is all brought into question because it relies on the use of the ordinary catheter or stiff stomach tube, which

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1. References to the literature will be found at the end of the article.

does not permit accurate quantitative studies. A still more serious criticism, and one which leaves the whole subject open, is that in none of the quoted work is the possibility of a continuous secretion of gastric juice sufficiently taken into account.

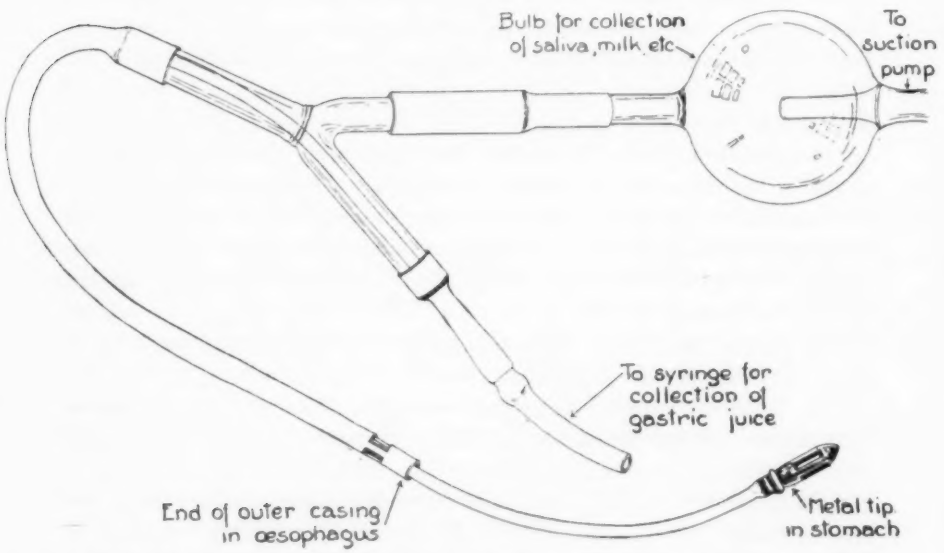
In 1888 Leo found free hydrochloric acid in the stomachs of unfed new-born babes, and noted that in older infants the stomach was rarely entirely empty, so that he could usually recover a few drops of thick, yellowish acid fluid. He washed out the stomach and again inserted the tube and then obtained only wash water from the preceding washings. Consequently Leo concluded that the acid juice obtained by him from the "empty" stomach was the gastric juice remaining from the last meal, concentrated by the absorption of water. Wohlmann reported that the secretion of the infant's empty stomach is viscid, colorless, glassy, and without free hydrochloric acid. Wohlmann took his specimens from one to two hours after feeding. The teachings of Pawlow that gastric secretion depends on appetite or on food or other stimuli in the stomach impressed the medical mind so deeply that until the present decade all gastric secretion was interpreted in the light of his investigations. A. H. Meyer concluded that the passage of the stomach tube does not excite the secretion of acid gastric juice. Pawlow's published work supports the same conclusion. Engel reports a 4-week-old babe with pyloric stenosis and a jejunal fistula. From this infant, who was fed through the fistula, Engel obtained by way of the esophagus from 60 to 200 c.c. of gastric juice daily. The total acidity of this juice ranged from 60 to 70 and was nearly entirely made up of free hydrochloric acid. Engel was unable to explain his findings except on the basis of a pathologic hypersecretion, which he thought might have caused the pyloric stenosis. Alfred F. Hess, in 1913, showed that the stomach of the unfed new-born babe secretes a highly acid juice, and he concluded further that saliva does not act as a stimulus to the production of such juice. He was unable to determine a relationship between the amount of sucking and the amount of juice secreted. Sedgwick recovered acid stomach and duodenal contents three and four hours after nursing. In 1905 Boldyreff reported continuous secretion of the gastric glands in starving dogs. Ten years later Fowler, Rehfuss and Hawk concluded that, in man, the gastric glands are never idle, while Carlson demonstrated the continuous secretion of gastric juice in the empty stomach of normal adults. Referring to its secretion during the hunger state, Carlson calls it hunger juice.

It is evident that the determination of the secretion of an appetite juice in the infant's stomach must be made in conjunction with the determination of its continuous secretion.

The flexible tube with the slotted weight at the tip described by Rehfuss, combined with any simple syringe for gentle aspiration, makes

an excellent instrument for the study of the physiology of the stomach of the infant. A smaller tip can be made for those infants who cannot swallow the ordinary tip. With this apparatus I have repeatedly recovered from the infant's stomach the entire 30 to 50 c.c. of water introduced into it and never have lost more than 2 c.c. in the washing. Furthermore, large, thick, gelatinous clumps of mucus and curd are removed without difficulty.

In order to avoid, as far as possible, contaminating the gastric juice with saliva, and to permit the carrying out of sham feeding, I converted a No. 21 F. soft rubber catheter into an outer casing for the Rehfuss tube. When in place this outer casing terminates internally in the esophagus, and externally with a suction apparatus. The whole is explained in the accompanying illustration, which is one-half actual size.


Bulb for collection of saliva, milk, etc.
To suction pump
To syringe for collection of gastric juice
End of outer casing in œsophagus
Metal tip in stomach

Author's apparatus for removing gastric contents from infants; one-half actual size.

The experimental procedure was as follows: If the babe fasted all night, he was given water at 5 a. m. in quantity equivalent to his usual feeding. When the stomach was examined a few hours later, milk remains were never found. If the period without food were shorter, his stomach was thoroughly washed out and observations begun an hour later.

If no aspiration is applied to the stomach tube during the half hour, the amount obtained is usually less than 1 c.c. The usual procedure was to insert the tube, exert suction to empty the stomach of any content, then allow the tube to remain one-half hour without suction, and

collect the specimen, if any. Repeat the procedure, exerting gentle suction every two and one-half minutes and collect the specimen. Exert suction in the same way during a third half hour while the sham feeding progresses. The final two specimens only are listed in the accompanying table.

As a rule, no secretion was obtained for five minutes after the insertion of the tube. On one occasion gastric juice containing free hydrochloric acid was obtained within two minutes of the time at which introduction of the tube began (Baby A.). This is less than the latent time usually required by the gastric glands (Carlson, Pawlow) and is further evidence that the secretion here obtained was not produced artificially by the apparatus.

To stimulate an appetite secretion, the babe was given a pacifier threaded over the tube, or, the food to which he was accustomed was administered by a medicine dropper, or, with the artificially fed babes, from their usual nursing bottle. The infant always sucked vigorously during this procedure. If the babe sucked before sham feeding began, it has been noted in the table. As a rule, the babes slept or were quiet and did not suck, except after the beginning of the sham feeding. The presence of the tube seemed to discommode these babes very little. There certainly was no psychic excitement to depress the action of the gastric glands while the babes were smacking and sucking over their food.

In three cases only, as noted in the table, did food reach the stomach. Strictly speaking, neither these instances nor the specimens which contained blood should be considered as offering evidence on either the subject of "hunger" or "appetite" gastric juice. The only demonstrable effect of the blood, which was never present in more than a trace, was to lower the acid titration values. On the three occasions on which milk reached the stomach, larger amounts of secretion was obtained.

The titrations were done against tenth-normal sodium hydroxid, using di-methyl-amino-azobenzol and phenolphthalein as indicators. The hydrogen-ion concentrations were done by the gas chain method.

I wish to thank Dr. J. F. McClendon for his courtesy in allowing the use of his apparatus.

The "appetite" gastric juice is characterized by its relatively profuse secretion and high acidity. Neither characteristic was present in the juice obtained after sham feeding in these infants. On the contrary, the juice obtained differed little in character and quantity from that obtained before sham feeding was begun.

It will be seen that the empty stomach of the infant continuously secretes a juice which at times is as acid as that of the adult, and that the infant's stomach does not secrete an "appetite" or psychic juice.

TABLE 1.—Comparative Secretion of Hunger and Appetite. Gastric Juice

Name	Age	Length of Preceding Starvation, Hours	Behavior During Collection of Continuous Secretion or "Hunger" Juice	Amount of "Hunger" Juice per ½ Hour, C.c.	Description of "Hunger" Juice	Method of Provoking Appetite Juice	Amount of Appetite Juice per ½ Hour, C.c.	Description of Appetite Juice
Ow.	15 hrs.	Unfed	Cried some; slept most of time	0.9	Slightly viscid; trace of saliva; no blood; translucent; free HCl 0; total acid 4			
Wal.	7 days	4	Slept.....	3.0	Thick, viscid trace of saliva; free HCl 8; total acid 18	Sucking on pacifier.....	3.0	Thick, viscid, trace of saliva; free HCl 2; total acid 18
Wal.	9 days	6	Quiet; no sucking...	3.0	Trace of saliva; free HCl 2.5; total acid 17.5			
Wes.	2 hrs.	Unfed	No sucking.....	6.0	$CH+ = 0.3 \times 10^{-2}$; free HCl 50; total acid 60			
Wes.	7 days	13	No sucking.....	1.75	Yellowish, clear, viscid; Gmelin test neg.; free HCl 18; total acid 39			
He.	6 days	11	Slept throughout; sucked a little	1.0	Slightly viscid; no saliva; no blood; free HCl 5; total acid 40			
P.	9 days	12	Slept; no sucking...	1.0	Viscid, clear, no foam; trace of blood; free HCl neg.; total acid 30	Sham feeding: 5 c.c. breast milk; sucked vigorously for 15 minutes	0.5	Thick, viscid, trace of blood; free HCl 0; total acid 30
A.	11 days	12	Slept most of time; no sucking	1.5	Slightly viscid trace of blood; free HCl 60; total acid 80	8 c.c. breast milk; sucked vigorously for 20 minutes	2.5	Brownish, blood stained, slightly viscid; free HCl 54; total acid 60
S.	12 days	13	Cried; no sucking...	2.0	Transparent, slightly viscid; free HCl 55; total acid 75	5 c.c. breast milk; sucked vigorously for 15 minutes	1.0	Thin, transparent, slightly viscid; free HCl 60; total acid 80
H.	17 days	13	Slept most of time; sucked occasionally	2.0	Clear, slightly viscid; trace of blood; free HCl 30; total acid 60	10 c.c. breast milk; sucked on tube	1.0	Clear trace of blood; free HCl 10; total acid 40
K.	3.5 mos.	17	Sucked on tube.....	3.5	Translucent viscid; trace of saliva; free HCl 7; total acid 28	5 c.c. cow's milk + saccharose; sucked	10.0	Clear, viscid, trace of saliva; small amount of milk; free HCl 0
K.	3.5 mos.	17	Sucked constantly	3.0	Trace of saliva; free HCl 33; total acid 38	Cow's milk + saccharose; sucked	2.0	Total acid 24; turbid, blood stained; free HCl 25; total acid 55
Ad.	5 mos.	16	Slept; no sucking...	3.0	Clear viscid, trace of saliva; free HCl 56; total acid 70	Malt soup	8.0	Contained milk; free HCl 18; total acid 45
Ad.	5 mos.	17	Slept; no sucking...	3.0	Thin, clear, slightly greenish; free MCl 80; total acid 110; $CH+ = 0.5 \times 10^{-1}$	15 c.c. malt soup; sucked vigorously 20 minutes	5.0	Contains 0.5 c.c. milk clot; free HCl 10; total acid 45; $CH+ = 2 \times 10^{-1}$
Wi.	7 days	25	Cried much; did not suck	1.0	Thick, viscid trace of blood; free HCl 10; total acid 40	Sham feeding: 10 c.c. breast milk; sucked vigorously; quiet	0.2	Clear mucus; free HCl 0
Ca.	9 days	24	Slept throughout; did not suck	3.0	Turbid, viscid fluid containing trace of blood; free HCl 12; total acid 32	Sham feeding: 10 c.c. breast milk; sucked vigorously; did not cry	0.9	Turbid, viscid, containing partially digested blood; free HCl 22; total acid 55
Ni.	11 days	23	Cried ⅓ of time; sucked a little	1.5	Viscid mucus; free HCl 0; total acid 8	Sham feeding: 10 c.c. breast milk; sucked vigorously; cried a little	1.0	Turbid, viscid, mucus; free HCl 0; total acid 10
P.	1 mo.	25	Slept ½ of time; cried a good deal; no sucking	1.8	Trace of saliva; viscid; free HCl +; total acid 9	Sham feeding: 10 c.c. breast milk; sucked some; cried much	1.0	Viscid mucus; trace of blood; free HCl 10; total acid 16
Wa.	5 mo.	21	Slept most of time; sucked a little	8.0	Clear, slightly viscid; free HCl 72; total acid 90	Sham feeding: 10 c.c. malt soup; sucked vigorously; whined and teased	14.0	Slightly viscid, cloudy; small amount of blood-stained mucus; free HCl 60; total acid 80

This accords with the absence of psychic inhibition of the hunger contractions.

As indicated by the digestion of egg white in Mett's tube, the infant's hunger juice contains pepsin.

Reiche has demonstrated the absence of a duodenal reflux into the infant's stomach. The present findings support his conclusion. What, then, becomes of this continuous secretion under circumstances such as enforced therapeutic starvation from twenty-four to forty-eight hours? Pfaundler has conjectured that at the close of digestion the alkaline secretion of the pyloric glands gradually neutralizes the acid content of the stomach. I cannot support this view. The finding of a greater quantity of juice when a more continuous suction is maintained, the frequent absence of juice when the tube is first inserted, and Sedgwick's finding that the young infant's duodenal contents are acid, favor the conclusion that at least a portion of this juice makes its way into the intestine.

It seems probable, therefore, that the secretion of the alkaline pancreatic and intestinal juices, which in the adult regurgitate into the stomach, as demonstrated by Boldyreff, and lower the acidity of the juice in the stomach (Carlson, Rehfuss and Hawk and Boldyreff), is, in the infant, relatively deficient.

The hunger juice is delivered through the tube intermittently. The most profuse secretion is, as a rule, associated with the higher acidities; this is also true in the adult (Carlson). The largest amounts were obtained from one of the unfed new-born babes and from the older infants. It is readily seen that the stomach of the starving infant can secrete from 50 to 200 c.c., or more, of highly acid juice daily. This equals the amount Engel obtained from his case of pyloric stenosis, which has served as the clinical basis for the theory that hyperacidity or hypersecretion of the gastric juice is an etiologic factor in that disease.

Furthermore, this demonstration of the capacity of the infant's stomach to secrete a highly acid juice, makes it probable that the low acid values found during gastric digestion of milk are in part due to its binding power for acid (Aron), and in part due to the relatively slight stimulation which it exerts on the gastric glands (Pawlow, Moore and Allanson). Huenekens found a hydrogen ion concentration of 174×10^{-5} in a 9½-months-old infant after a meal of soup and vegetables. Most of his results were lower, however. No such studies have been made in younger infants.

Experience in the clinic of the University of Minnesota and in other clinics (Rott) has proved the advantage which is gained in feeding the premature infant by tube. Theoretical objections to the use of

the tube have been based principally on the assumed existence of an appetite gastric juice (Pfaundler).

The amount of saliva collected during the experiments on gastric secretion was measured in six cases.

W.— 7 days	No sham feeding	7 c.c. in 40 minutes
H.—17 days	Sham feeding	14 c.c. in 2 hours
P.— 1 mo.	Sham feeding	25 c.c. in 1.5 hours
Ne.—11 days	Sham feeding	15 c.c. in 1.5 hours
P.— 9 days	Sham feeding	8 c.c. in 1.5 hours
S.—12 days	Sham feeding	10 c.c. in 1.5 hours

The saliva collected was the thick viscid product of the submaxillary glands, which Schilling has noted as being preponderant during early infancy.

Allaria points out the chemical and mechanical advantages of having the milk well mixed with saliva, and estimates that the infant secretes an amount equal to from 10 to 20 per cent. of the ingested food. The tube-fed infant may do without this secretion in part or altogether, but there is no evidence that his gastric secretion is less than that of the actively nursing babe.

What light does this study throw on deprivation of food as a therapeutic agent? In infancy such a measure finds its chief field in acute alimentary disorders and summer diarrheas. The significant fact is that in hunger the infant's stomach secretes continuously, but with intermittent intensity, a highly acid juice, which at least in part flows into the small intestine where it may play a disinfecting or detoxicating rôle.

SUMMARY

1. Description of an apparatus by which sham feeding can be carried out and gastric juice collected under conditions which give positive evidence of the amount secreted.

2. There is no appetite or psychic secretion of gastric juice in the young infant. This disproves the present view, which is based on insufficient experimental evidence.

3. The empty stomach of the hungry babe secretes a gastric juice which often is as acid as that found in the adult's stomach.

4. The more profuse this secretion, the higher is its acidity. It contains pepsin.

5. This secretion is not neutralized in the stomach, but flows out into the small intestine. Regurgitation through the infant's pylorus does not occur.

6. The theoretical objections to tube feeding in prematures because of the lack of stimulation of an appetite gastric juice are not valid. However, a disadvantage may lie in this: that such feeding precludes the usual admixture of the milk with saliva.

Therapeutic starvation in acute alimentary disorders and in summer diarrheas may owe its success in part to the heightened tonus of the alimentary tract, and in part to the pouring out of highly acid detoxicating and disinfecting gastric juice into the small intestine.

I wish to acknowledge my indebtedness to Dr. J. P. Sedgwick for the use of material from his service in the University of Minnesota Hospital.

REFERENCES

1. Allaria, G. B.: Ueber die Wirkung des Speichels im Anfangsstadium der Verdauung beim Säugling. Jahrb. f. Kinderh., 1911, **74,** 252; ibid., Die Wirkung des Speichels beim Säugling auf die Cohäsionskraft des Labgerinnsels. Pediatria, 1911, **19,** 10. Abstr. in Arch. f. Kinderh., 1911, **59,** 129; ibid., Quanta saliva secerne il lattante durante la poppata? Riv. di clin. pediat., 1912, **10,** 439.

2. Aron, H.: Das Salzäurebindungsvermögen von Frauen und Kuhmilch. Jahrb. f. Kinderh., 1914, **79,** 288.

3. Bauer, L., and Deutsch, E.: Das Verhalten der Magensäure, Motilität und Resorption bei Säuglingen und Kindern unter psysiologischen und pathologischen Verhältnissen. Jahrb. f. Kinderh., 1898, **48,** 21.

4. Bogen: Experimentelle Untersuchungen über psychische und assoziative Magensaftsekretion beim Menschen. München. med. Wchnschr., 1907, **54,** 812.

5. Boldyreff, W.: The Self-Regulation of the Acidity of the Gastric Contents and the Real Acidity of the Gastric Juice. Quart. Jour. Exper. Physiol., 1914, **7,** 1.

6. Boldyreff, W.: Ueber der Uebergang des natürlichen Mischung des Pankreas, des Darmsaftes und der Galle in den Magen. Centralbl. f. Physiol., 1904, **18,** 457.

7. Bönniger, M.: Magenfunktion und Psyche. Deutsch. med. Wchnschr., 1913, **39,** 890.

8. Cohnheim, O., and Soetbeer, F.: Die Magensaftsecretion des Neugeborenen. Ztschr. f. physiol. Chem., 1902-1903, **37,** 467.

9. Engel: Zur Kenntnis der Magensaftsecretion beim Säugling. Arch. f. Kinderh., 1908, **49,** 16.

10. Fowler, C. C., Rehfuss, M. E., and Hawk, P. B.: An Investigation of the Gastric Residuum in Over One Hundred Normal Cases. Jour. Am. Med. Assn., 1915, **65,** 1021.

11. Hess, A. F.: The Gastric Secretion of Infants at Birth. Am. Jour. Dis. Child., 1913, **6,** 264.

12. Huenekens, E. J.: Die Acidität des Mageninhalts im Säuglings und Kindesalter bei Milch und fleischhaltiger Probenahrung. Ztschr. f. Kinderh., 1914, **11,** 297.

13. Leo, H.: Ueber die Function des normalen und kranken Magens und die therapeutischen Erfolge der Magenausspülung in Säuglingsalter. Berl. klin. Wchnschr., 1888, **25,** 981.

14. Meisl, A.: Ueber die Beziehungen zwischen Appetit und Magensaftsekretion. Wien. klin. Rundschau., 1907, No. 49. Quoted by Uffenheimer: Ergebn. d. inn. Med. u. Kinderh., 1907, **2,** 271.

15. Meyer, A. H.: Zur Kenntniss der Magensaftsecretion der Säuglings. Arch. f. Kinderh., 1902, **35,** 79 and 177.

16. Moore, C., and Allanson, H. E.: Influence of Foodstuffs on Gastric Secretion. Brit. Med. Jour., 1911, **2,** 1535.

17. Nothmann, H.: Zur Frage der psychischen Magensaftsekretion beim Säugling. Arch. f. Kinderh., 1909, **51,** 123.

18. Pawlow, I. P.: The Work of the Digestive Glands. Trans. by W. H. Thompson. Ed. 2, London, Griffin, 1910, p. 90.

19. Pfaundler: Verhandl. d. Cong. f. inn. Med., Wiesbaden, 1899, p. 337; quoted by Czerny and Keller: Des Kindes Ernährung, p. 62.

20. Rehfuss, M. E., and Hawk, P. B.: Direct Evidence of the Secretion of a Gastric Juice of Constant Acid Concentration by the Human Subject. Jour. Am. Med. Assn., 1914, **63,** 2088.

21. Reiche, A.: Zur Frage des Rückflusses vom Pankreassaft in den Magen des Säuglings. Ztschr. f. Kinderh., 1913, **6,** 235.

22. Schilling, F.: Zur Sekretion der Speicheldrüsen; insbesondere der Glandula submaxillaris, im Säuglinsalter. Jahrb. f. Kinderh., 1903, **58,** 518.

23. Sedgwick, J. P.: The Hydrogen Ion Concentration of the Gastric and Duodenal Contents in Childhood. Tr. Am. Pediat. Soc., 1915, p. 39.

24. Schmidt, A. Von: Brustsaugen und Flaschensaugen. München. med. Wchnschr., 1904, **51,** 2141.

25. Wohlmann, L.: Ueber die Salzäureproduktion des Säuglingsmagnens im gesunden und kranken Zustande. Jahrb. f. Kinderh., 1891, **32,** 297.

STUDIES IN THE NEPHRITIS OF CHILDREN*

FIRST PAPER

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RENAL FUNCTION TESTS

For many years the study of nephritis has attracted a great deal of attention from clinicians. The problems involved are so complicated and so many, the correlation of different anatomic lesions with variations in functional capacity is so difficult, and the stages through which any given case of nephritis may pass are sometimes so varied in the pictures they present, that it is indeed impossible for anyone to feel that he understands nephritis, no matter how much experience he may have had with it. Up to ten or twelve years ago the problem that chiefly interested investigators was a study of the histologic changes produced in the kidney tissue by different types of nephritis, and little attention was paid to changes in the functional capacity of the organ. At present the reverse is true, and studies in kidney function dominate the literature on nephritis in the last decade. Nearly all the work, however, has been done with adults, and nephritis in children has received comparatively little attention.[1] Two years ago, at the suggestion of Dr. John Lovett Morse, I became interested in nephritis, and since then have studied between sixty and seventy cases of acute and chronic nephritis in the wards of the Children's Hospital. During the course of these investigations many interesting questions have arisen, which will be discussed later, and the present paper will take up only functional tests.

There is this great difference between nephritis as seen in adults and in children. In adults there may be recognized, broadly speaking, two great groups of nephritic cases; one dependent on acute infectious processes somewhere in the body, the toxins of which injure the kidneys either temporarily or permanently; the other dependent on slow-going degenerative changes in the blood vessels, which produce so-called "chronic interstitial" nephritis, or "cardiorenal" disease. By far the majority of cases of nephritis in adults fall into the second group, and

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1. For a review of the literature dealing with nephritis in children see Hill, L. W.: Boston Med. and Surg. Jour., 1917, **177**, 313.

it is this group of cases which has been most extensively studied. On the other hand, I do not feel that it is too strong to say that nearly all nephritis in children depends on infection; for practical purposes we can rule out the slow-going "vascular" or chronic interstitial type. (A few cases of "chronic interstitial" nephritis in children have been reported, however.) Thus it may be seen at the outset that in dealing with nephritis in children we are dealing with a far different problem from that of nephritis in older persons, and we must not necessarily expect functional tests to show the same phenomena as in the adult.

Many tests of kidney function have been used in the last few years, some of which have passed into oblivion, some of which have survived. These functional tests may be divided into two broad groups, the first of which comprises those tests that measure the power of the kidney to excrete chemical substances not ordinarily contained in the food, or "körper fremden" substances, as the Germans have put it. The second group comprises those tests which measure the ability of the kidney to excrete substances ordinarily contained in the food. Three of the most important functions of the kidney are to excrete nitrogen, salt and water, and it is these, therefore, that are usually taken as the test substances to determine the kidney's functional power.

Phthalein Test.—A number of tests with substances foreign to the body have been proposed, but the only one which has survived is the phenolsulphonephthalein test of Rowntree and Geraghty. This has been found, after extended use all over the world, to be the most practical test of kidney function for general clinical use.

In this test, which is now familiar to everyone, 6 mg. of the dye phenolsulphonephthalein is injected intramuscularly, and all urine passed for two hours is saved. This is made alkaline with sodic hydrate to bring out the color of the phthalein, diluted to 1,000 c.c., and the intensity of the resulting color is compared with that of a standard solution. The result is expressed in per cent. of phthalein excreted in two hours; the test is therefore one of the rapidity of the kidneys to excrete the dye. A normal adult should excrete from 50 to 80 per cent. of the phthalein in two hours, and any figure above 50 is usually considered normal for an adult. The figures for children vary considerably from this, as will be seen.

The results of this test in twenty-seven normal children are shown in Table 1. These children might not perhaps be considered as strictly normal children, inasmuch as they were all in the hospital for various reasons, but they were all free from any condition which could possibly influence the function of the kidney, such as fever, malnutrition, etc. A Duboscq colorimeter was used.

It may then be said that the average phthalein excretion for normal children is somewhere in the neighborhood of 75 per cent., and that if the function is below 60 per cent. it may be considered as abnormal.

These figures agree fairly closely with those of Tileston and Comfort[2] and Leopold and Bernhard,[3] whose average figures for normal children are 78 per cent. to 81 per cent., and 70 per cent., respectively.

The test was performed on thirty-seven children with nephritis of all degrees of severity. Twenty-one cases were acute, sixteen chronic.

The average excretion of the twenty-one acute cases was 59 per cent., or 17 per cent. below the normal average; 59 per cent. would be a normal phthalein excretion for most adults.

The average excretion of the sixteen chronic cases was 63 per cent., or 13 per cent. below the normal average of 76 per cent.

TABLE 1.—PHENOLSULPHONEPHTHALEIN EXCRETION IN NORMAL CHILDREN

Age, Years	Phthalein Excretion, Per Cent.	Age, Years	Phthalein Excretion, Per Cent.
2½	64	8	77
5	77	8	79
6	80	9	66
6	71	9	70
7	69	10	80
7	90	10	74
7	77	10	65
7	77	10	100
7	66	10	76
7	66	11	80
7	74	11	77
7	80	11	80
8	87	11	90
8	87		

High, 100 per cent.; low, 64 per cent.; average, 76 per cent.

DISCUSSION

From the figures in Table 2 it can be said that the phthalein excretion is diminished in many cases of acute or of chronic nephritis in children, and that it is likely to be a little lower in the acute cases than it is in the chronic. The reverse is true in adults. Many chronic "cardiorenal" cases show a phthalein excretion of 5 to 10 per cent., or even sometimes such a slight trace that it cannot be read at all. Most cases of chronic nephritis in children are of a mild type, and even the very severe cases, of which we have seen a number, do not show nearly as much interference with phthalein excretion as one would expect. It is no uncommon thing to see a normal excretion in a child who has had nephritis for two or more years, who is edematous and anemic, and who has an increased blood pressure and a urine loaded with albumin and casts. In dealing with children, then, normal or high phthalein excretion helps us very little, as the test may be high in many cases in which there is unquestionably severe kidney damage. A low

2. AM. JOUR. DIS. CHILD., 1915, **10,** 278.
3. AM. JOUR. DIS. CHILD., 1916, **11,** 432.

TABLE 2.—PHENOLSULPHONEPHTHALEIN EXCRETION IN NEPHRITIS

ACUTE CASES

Age, Years	Phthalein Excretion, Per Cent.	Remarks
9	63	Mild
9	62	Severe; bloody urine; blood pressure 140
6	62	Severe; bloody urine
4	80	Edematous; blood pressure 135
10	54	Moderately severe
8	50	Moderately severe; considerable edema
5	66	Moderately severe
10	71*	Severe; much edema
5	76	Moderately severe; slight edema
3	53, 50	Moderately severe; slight edema
4½	66	Mild
5	65	Moderately severe
3	83	Moderately severe
4	30	Moderately severe
5	70	Mild
7	64, 35	Severe; much edema
8	43	Moderately severe
4	43	Moderately severe
4	75	Severe; much edema
4	21	Severe; much edema
5	43	Severe; much edema

CHRONIC CASES

7	55	Chronic interstitial nephritis; blood pressure 210
9	70	Moderately severe; two years' duration; blood pressure 150
7	66	Mild; one year's duration
9	80	Mild; subacute
2	66	Very severe; much edema; blood pressure 130
12	66	Mild
11	69	Very severe; much edema
7	50, 37, 70	Moderately severe; one year's duration
5	33, 60, 54, 30, 44	Very severe; two years' duration
6	62, 71, 60	Severe
4	87	Mild; subacute
6	50	Moderately severe
13	71	Mild; two years' duration
4	50	Moderately severe
3	60	Moderately severe
8	59	Moderately severe

*After decapsulation.

The average excretion of the twenty-one acute cases was 59 per cent., or 17 per cent. below the normal average; 59 per cent. would be a normal phthalein excretion for most adults.

function, however, tells us a good deal: in acute nephritis it puts us on our guard, and makes us consider the case a severe one whether or not it appears so clinically; in chronic nephritis it tells us that permanent damage has been done to the kidney, and makes us very guarded in our prognosis.

Added Salt and Urea Test.—Another method of testing renal function which has been in vogue is the addition of a known amount of salt and urea to a standard diet, and then determining how much of the

added salt and urea has been excreted in a given period of time. In adults this test is usually performed as follows:[4]

> The patient is put on a constant standard diet containing a known amount of nitrogen, salt and water, and the amounts of salt and nitrogen in the urine are quantitated daily until their excretion has become constant. This usually takes from two to three days. On the third or fourth day 10 gm. of salt is added to the diet and by daily urine analyses it is determined how long the kidneys take to excrete the added salt. Normally it should be excreted in two days. After the ability of the kidneys to excrete salt has been determined, 20 gm. of urea is added to the diet, and the rapidity of excretion of this is determined in the same way.
>
> This test necessitates keeping the patient on a carefully fixed diet and collecting accurate twenty-four hour urine specimens for eight or ten days. Any such procedure as this is obviously impossible in the case of small children, owing to refusal to eat, wetting the bed, etc. It was felt desirable to modify this test in dealing with children, and this was done as follows:
>
> For three days the child was kept on the following standard diet:
>
> | | |
> |---|---|
> | Oatmeal, 4 tablespoonfuls | 1 orange |
> | Bread, 3 slices | 1 small baked potato |
> | 16 per cent. cream, 4 ounces | Sugar, 4 drams |
> | Salt-free butter, 2 ounces | Water, 34 ounces |
>
> Whole milk, 16 ounces
>
> This diet contains about 5.7 gm. of nitrogen. On the third day the twenty-four-hour output of salt and nitrogen in the urine was determined, and the next day 10 gm. of urea (containing 4.65 gm. nitrogen) and 5 gm. of salt were added to the diet. (Given together in a little water, early in the morning.)
>
> The nitrogen and salt were then quantitated in the following twenty-four hour urine specimen, and by subtracting the urinary nitrogen and salt of the previous day the amount of these substances eliminated in the twenty-four hours after the addition was determined. I realize that it is better to give the salt and urea on different days than to give them together, and that there are also other objections to this simplified method of procedure, but even with this simple method many failures resulted, owing to th: capriciousness of the child's appetite, unwillingness to take the salt and urea, wetting the bed, etc., and the cases reported represent only a portion of many attempted.

Twelve cases of nephritis were studied by this method, six of them acute, six chronic.

DISCUSSION

As may be seen from a glance at the table of normals (Table 3), the salt and urea excretion varied normally within rather wide limits, and it is therefore difficult to establish a normal standard. Age seemed to bear no relation to a large or a small excretion. Inasmuch as normal children vary so much in their reaction to this test, it is valuable only within very wide limits, and the excretion of salt and nitrogen may be said to be abnormal only if it is very much impaired. In six of the twelve cases studied the excretion of salt, of urea, or of both, may be said to be abnormally low (those marked †); in the rest of the cases it may be said to be normal. Marked impairment of salt excretion

4. Frothingham: Am. Jour. Med. Sc., 1915, **149**, 808.

occurred more frequently than of nitrogen, especially in those cases which were, or had been, edematous.

Value of the Test.—The added salt and urea test may be of some value in indicating that the function of the kidneys is impaired, but only if the excretion is very poor, inasmuch as the normal response to the test may vary within such wide limits. If the excretion of both salt and urea is high, it is safe to assume that the kidney's function is

TABLE 3.—Results of Salt and Urea Test of Renal Function

Age, Years	Salt and Nitrogen Above Control * Salt, Gm.	Nitrogen, Gm.	Remarks
NORMALS			
11½	4.24	3.33	
10	1.47	5.00	
8	2.90	3.60	
10	2.40	3.40	
6	3.16	4.12	
11	4.22	4.32	
6	2.16	3.70	
6	3.78	4.55	
NEPHRITICS			
4	—0.25	1.96	Severe acute nephritis; no edema†
4	2.96	2.80	Mild acute nephritis; slight edema
10	3.84	2.88	Mild acute nephritis; no edema
7	1.90	3.24	Severe acute nephritis; no edema
8	3.90	3.16	Severe acute nephritis; no edema
9	3.80	3.00	Moderately severe acute nephritis; no edema
4	1.37	2.53	Severe chronic nephritis; considerable edema
9	—1.60	—2.86	Severe chronic nephritis; moderate edema†
7	3.02	2.40	Mild chronic nephritis
7	4.88	5.62	Mild subacute nephritis
11	4.22	6.26	Mild chronic nephritis
6	5.45	3.85	Severe chronic nephritis after decapsulation (Edebohls' operation)

* By this is meant the excess of salt and nitrogen excreted on the test day over that of the previous day.

† A minus sign indicates that less salt or urea were excreted on the day after their addition to the diet than on the day before.

Twelve cases of nephritis were studied by this method, six of them acute, six chronic.

not much impaired, as the test is an extremely severe one, for Zondek[5] has shown that diseased kidneys may be able to eliminate a large amount of urea or of salt *alone,* but not a large amount of both substances when they are ingested together. Even if this test occasionally does yield information of value, it is not at all adapted for general use in children, as the difficulties of carrying it out accurately are well nigh insurmountable.

5. Zondek: Ztschr. f. klin. Med., 1915, **82,** Nos. 1 and 2.

The "Two Hour" Renal Test.—This test was first proposed by Hedinger and Schlayer[6] in 1914. In this country it has been used extensively by Mosenthal[7] in Baltimore, and O'Hare[8] in Boston. It is performed as follows:

> The patient is put on a full diet, containing a considerable amount of protein, of purin extractives and of salt, in order to make a demand on the power of the kidneys to excrete such a diet. The fluid allowance is fixed, and no fluid is allowed between meals. The noon meal is considerably larger than either of the others and is designed particularly to call forth the kidney's power of excretion. The urine during the test day is collected in two-hourly periods, and the night urine in one sample. The amount and specific gravity of each two hourly specimen is noted as well as the amount and gravity of the night urine. The amount of salt and nitrogen and the salt and nitrogen concentration of each specimen are quantitated.

Mosenthal[7] has well explained the principles of this test as follows:

> The kidney expresses its diminished power to functionate by a fixation of its power of concentration. The normal kidney yields a urine of medium, low or high specific gravity according to the proportion of fluids and solids that must be excreted in order to maintain the composition of the body fluids at a constant level. The diseased kidney, on the other hand, loses this flexibility, and the power to answer the demand for a more concentrated or a more dilute urine no longer exists.

The normal response to a test meal is shown by a considerable variation in the amount, specific gravity, nitrogen and salt concentration of the two-hourly specimens, and by a relatively small amount of night urine, of high (1.020) specific gravity, and high nitrogen concentration (1 per cent.). A kidney whose function is impaired excretes a urine which varies very little in specific gravity and nitrogen and salt concentration in each two-hourly period. The gravity may be fixed at a low or a fairly high level. Also the amount of night urine is likely to be large, and the nitrogen concentration and specific gravity low (below 1 per cent. and 1.020, respectively). The fixation of the specific gravity is the most important point to observe, and it is not at all necessary to make salt or nitrogen determinations on any of the specimens. Moreover, it is not essential that the patient consume the exact diet indicated; it is merely necessary that the diet be a full one, containing a considerable amount of purin bases, nitrogen and salt, and that the noon meal shall contain more of these than the other meals. It is essential that the fluid taken with each meal be the same in amount, and that no fluids be taken between meals or at night. The test takes only one day, does not require the patient to be on a particularly rigid diet, and if, perhaps, one two-hour urine specimen is lost, no particular harm is done. *This, therefore, is a test which is very well adapted for ordinary clinical use, as in its simplest form no long drawn out procedure or complicated urinary analyses are necessary.*

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6. Schlayer: Arch. f. klin. Med., 1914, **114,** 120.
7. Mosenthal: Archives Int. Med., 1915, **16,** 733.
8. O'Hare: The Archives Int. Med., 1916, **17,** 711.

I have modified the original procedure somewhat, especially for use in children, and have carried it out in this manner.

The child is put on the following diet:

Breakfast	Dinner
Cereal, 2 tablespoonfuls	Chopped meat, 2 tablespoonfuls
Bread, 1 slice	1 egg
Butter, ½ square	1 potato
Apple sauce, 2 tablespoonfuls	Butter, 1½ cubes
Milk, 6 ounces	Milk, 6 ounces
Water, 4 ounces	Water, 4 ounces
Extra salt, 1 gm.	Extra salt, 1 gm.
Caffein sodium benzoate, 2 grains	Caffein sodium benzoate, 2 grains

The caffein is given instead of the tea or coffee which is given to adults to secure diuretic response from the kidneys.

The two-hour urine specimens are then collected in the usual manner, starting at 6 in the morning, and continuing until 6 at night. The night urine is collected from 6 p. m. to 6 a. m.

Then the gravity of each two-hourly specimen, and the amount of night urine is measured, a fixation of gravity in each specimen indicating loss of power on the part of the kidney to vary the concentration of solids in the urine, and hence impaired function. The determination of the specific gravity of each specimen is really all that is essential, but in most cases we have measured the day and night urine, and in some the nitrogen concentration of the night urine.

As controls, fifteen normal children were studied, for as far as we know, no work with this test has ever been done with children, and it is desirable to establish a standard of normality.

NORMALS

Time	No. 1 Aged 11 Yrs. Specific Gravity	No. 2 Aged 10 Yrs. Specific Gravity	No. 3 Aged 11 Yrs. Specific Gravity
6- 8	1.018	1.013	1.016
8-10	1.010	1.021	1.022
10-12	1.015	1.023	1.023
12- 2	1.021	1.024	1.020
2- 4	1.017	1.027	1.015
4- 6	1.021	1.028	1.011
6- 6	1.023	1.020	1.020
	Night urine, 260 c.c.	Night urine, 320 c.c.; nitrogen concentration, 88%	Night urine, 165 c.c.; nitrogen concentration, 1.1%

Time	No. 4 Aged 7 Yrs. Specific Gravity	No. 5 Aged 10 Yrs. Specific Gravity	No. 6 Aged 10 Yrs. Specific Gravity
6- 8	1.017	1.016	1.015
8-10	1.025	1.009	1.016
10-12	1.025	1.018	1.022
12- 2	1.022	1.009	1.028
2- 4	1.030	1.025	1.020
4- 6	1.026	1.025	1.025
6- 6	1.032	1.027	1.027
	Night urine, 75 c.c.; nitrogen concentration, 2%	Night urine, 210 c.c.; nitrogen concentration, 1.7%	Night urine, 325 c.c.

Time	No. 7 Aged 6 Yrs. Specific Gravity	No. 8 Aged 9 Yrs. Specific Gravity	No. 9 Aged 10 Yrs. Specific Gravity
6- 8	1.019	1.018	1.017
8-10	1.022	1.018	1.004
10-12	1.027	1.024	1.019
12- 2	1.027	1.028	1.020
2- 4	1.025	1.027	1.023
4- 6	1.028	1.026	1.025
6- 6	1.030	1.025	1.030
	Night urine, 160 c.c.; nitrogen concentration, 2.5%	Night urine, 480 c.c.; nitrogen concentration, 1.10%	Night urine, 120 c.c.

Time	No. 10 Aged 9 Yrs. Specific Gravity	No. 11 Aged 9 Yrs. Specific Gravity	No. 12 Aged 9 Yrs. Specific Gravity
6- 8	1.018	1.022	1.020
8-10	1.017	1.212	1.012
10-12	1.020	1.018	1.009
12- 2	1.015	1.018	1.020
2- 4	1.015	1.014	1.019
4- 6	1.023	1.017	1.016
6- 6	1.020	1.023	1.020
	Night urine, 350 c.c.; nitrogen concentration, 1.1%	Night urine, 290 c.c.	Night urine, 260 c.c.

Time	No. 13 Aged 6 Yrs. Specific Gravity	No. 14 Aged 6½ Yrs. Specific Gravity	No. 15 Aged 6 Yrs. Specific Gravity
6- 8	1.025	1.024	1.015
8-10	1.032	1.027	1.025
10-12	1.035	1.024	1.015
12- 2	1.026	1.020	1.020
2- 4	1.027	1.021	1.025
4- 6	1.029	1.028	1.020
6- 6	1.029	1.028	1.010
	Night urine, 70 c.c.	Night urine, 200 c.c.	

DISCUSSION OF NORMALS

Normal children show about the same response to the nephritic test meal that normal adults do. It will be noted as one looks over the results obtained in the normal cases that there is considerable variation in the gravity of the two-hour specimens in each case. *In every normal case there is a difference of at least 8 points between the highest and lowest gravity of the two-hour specimens, and usually there is a difference considerably greater than this.* These tables show very well how in health the concentration of the urine is varied from hour to hour. In every normal case except one, the specific gravity of the night urine was over 1.020. The amount of night urine in the normals varied too much to be able to determine on any definite standard of normality. Sixteen nephritics were studied. These were various types of cases, some acute, some subacute or chronic, some mild, others severe. In most cases the phthalein test was made in addition to the test meal, for purposes of comparison, and in a few the determination of the concentration of blood urea nitrogen. No case was studied which was

edematous, as conditions of urinary excretion are changed in such a way by edema that the two-hour renal test does not apply to this group of cases.

ACUTE NEPHRITIS

CASE 1.—R. M., aged 6 years. Severe acute nephritis. Urine: smoky; albumin, large trace; sediment shows many hyaline and granular casts, and many red blood cells. Time of test, one month after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.018	2- 4	1.018
8-10	1.020	4- 6	1.018
10-12	1.020	6- 6	1.022
12- 2	1.018		

Blood urea nitrogen = 21 mg. per 100 c.c. blood.

Night urine 40 c.c.

Interpretation: Fixation of gravity at a moderately high level, characteristic of acute nephritis in the stage of repair; indicates that normal function has not yet returned.

CASE 2.—H. K., aged 3⅔ years. Severe acute nephritis. Urine: albumin, trace; sediment shows many red blood cells and a rare cast. Phthalein excretion = 33 per cent. Time of test, three weeks after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.010	2- 4	1.011
8-10	1.014	4- 6	1.013
10-12	1.013	6- 6	1.013
12- 2	1.015		

Night urine, 180 c.c.; nitrogen concentration, 1.06 per cent.

Interpretation: Fixation of gravity at a low level; phthalein test lower than it should be at this stage, indicating the possible development of a chronic process; night urine of low gravity, but with a normal nitrogen concentration.

CASE 3.—J. C., aged 10 years. Severe acute nephritis. Urine: bright red; albumin, large trace; sediment shows many red blood cells, and a few bloody and granular casts. Blood urea nitrogen 32 mg. per 100 c.c. blood. Phthalein excretion = 40 per cent. Time of test, four days after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.012	2- 4	1.014
8-10	1.015	4- 6	1.015
10-12	1.015	6- 6	1.015
12- 2	.....		

Night urine, 505 c.c.; nitrogen concentration, 0.67 per cent.

Interpretation: Marked fixation of gravity at a low level; low phthalein test; moderately increased blood urea, indicating retention of nitrogen; a decided nocturnal polyuria. All tests indicate a considerable degree of functional impairment.

CASE 4.—J. M., aged 4 years. Severe acute nephritis. Urine: albumin, large trace; sediment shows many casts and red blood cells. Phthalein excretion = 75 per cent. Blood urea nitrogen = 19 mg. per 100 c.c. blood. Time of test, three weeks after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.020	2- 4	1.020
8-10	1.020	4- 6	1.022
10-12	1.022	6- 6	1.022
12- 2	1.021		

Night urine 202 c.c.

Interpretation: Fixation of gravity at a high level; normal blood urea

and phthalein; clinically a severe case of nephritis, but with high functional tests. Here the two-hour renal test indicates a damaged kidney when the other tests give no indication of it.

CASE 5.—L. N., aged 5 5/12 years. Moderately severe acute nephritis. Urine: albumin, absent; sediment shows a few casts. Time of test, 1 month after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.005	2- 4	1.020
8-10	1.017	4- 6	1.021
10-12	1.020	6- 6	1.025
12- 2	1.020		

Night urine, 200 c.c.; nitrogen concentration, 1.1 per cent.

Interpretation: No fixation of gravity; a normal response to the test meal; high gravity and nitrogen concentration of night urine. Tests indicate a kidney which has nearly recovered, and in which there is no chronic process developing.

CASE 6.—M. P., aged 5 1/3 years. Mild acute nephritis. Urine: albumin, none. Sediment shows rare hyaline and granular casts. Phthalein excretion = 83 per cent. Time of tests, seven weeks after onset; boy clinically well.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.025	2- 4	1.024
8-10	1.023	4- 6	1.022
10-12	1.020	6- 6	1.030
12- 2	1.020		

Night urine, 170 c.c.; nitrogen concentration, 1 per cent.

Interpretation: No fixation of gravity; high gravity and nitrogen concentration of night urine; high phthalein excretion. Tests show, in conjunction with his good general condition, that he has practically recovered, and is in no danger of developing a chronic condition.

CASE 7.—R. I., aged 8 years. Moderately severe acute nephritis. Urine: albumin, trace. Sediment shows large numbers of red blood cells and rare cast. Phthalein excretion = 43 per cent. Time of tests, three weeks after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.018	2- 4	1.015
8-10	1.018	4- 6	1.015
10-12	1.015	6- 6	1.018
12- 2	1.016		

Night urine, 230 c.c.; nitrogen concentration, 0.77 per cent.

Interpretation: Fixation of gravity at a low level; low concentration of night urine; low phthalein excretion. A damaged kidney, which still shows severe interference with function.

CASE 8.—S. B., aged 4 years. Acute nephritis. Urine: albumin, none. Sediment shows rare red blood cell. Boy clinically well. Phthalein excretion = 43 per cent. Time of tests, one month after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.014	2- 4	.....
8-10	1.012	4- 6	1.015
10-12	1.015	6- 6	1.018
12- 2	1.015		

Night urine, 340 c.c.; nitrogen concentration, 0.70 per cent.

Interpretation: Fixation of gravity at a low level; large quantity of night urine with a low nitrogen concentration; low phthalein excretion. Although the boy is clinically well, and shows no albumin in the urine and little in the sediment, the functional tests indicate that his kidneys are not functioning

normally. This means that it is possible he is developing a chronic process, and would tend to make us watch him rather carefully for the next few months.

CASE 9.—J. M., aged 10 years. Acute nephritis. Urine: albumin absent. Sediment shows a rare granular cast and a rare red blood cell. Time of test, three weeks after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.016	2- 4	1.011
8-10	1.010	4- 6	1.018
10-12	1.015	6- 6	1.020
12- 2	1.006		

Night urine 400 c.c.

Interpretation: No fixation of gravity; probably no impairment of function. This boy had a very marked response to the diuretic materials of the test meal, passing 1,260 c.c. of urine in the test twenty-four hours, with a fluid intake of only 900 c.c.

CASE 10.—J. W., aged 2⅝ years. Mild subacute nephritis. Urine: albumin absent. Sediment shows a rare red blood cell and a rare cast. Time of test, three months after onset.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.016	2- 4	.....
8-10	1.005	4- 6	1.020
10-12	1.015	6- 6	1.024
12- 2	1.014		

Night urine 160 c.c.

Interpretation: No fixation of gravity. The good general condition of the child, and the normal response to the test meal show that he is developing no chronic process, although his urine has contained a few blood cells for a long time.

CHRONIC CASES

CASE 11.—M. H., aged 11 years. Very mild chronic nephritis. Urine: albumin, slightest possible trace occasionally. Sometimes the urine contains none. The sediment sometimes shows a rare cast. Blood urea nitrogen, 14 mg. per 100 c.c. blood. Phthalein excretion = 70 per cent.

Time	Specific Gravity	Salt Concentration, Per Cent.
8-10	1.010	0.60
10-12	.....	....
12- 2	1.010	0.62
2- 4	.....	....
4- 6	1.020	0.70
6- 8	1.020	0.94
8- 8	1.021	0.88

Night urine, 315 c.c.; nitrogen concentration, 0.92 per cent.

Interpretation: No fixation of gravity; no fixation of salt concentration; reasonably high gravity and nitrogen concentration of night urine; normal blood urea nitrogen and normal phthalein excretion. Functional tests together indicate that the nephritic condition is extremely mild, probably nonprogressive, and that as far as can be told from such tests, the function of the kidney is normal.

It is in just this type of case that functional tests are of the most value. Here is a girl who has had an acute nephritis, and whose urine has not cleared up as it should, still containing a little albumin and casts occasionally. If we can determine by our functional tests that the function of the kidney is normal, it aids us immensely in prognosis and makes us feel that the urine

will eventually clear up, and that the girl will not have a seriously progressive nephritis.

CASE 12.—F. L., aged 5½ years. Chronic nephritis of two years' duration; clinically severe. Urine: albumin, trace. Sediment shows many hyaline and granular casts. Phthalein excretion = 25 per cent.

March 23, 1916		May 7, 1917		May 8, 1917
Time	Sp. Gr.	Time	Sp. Gr.	Sp. Gr.
7- 9.....	1.007	6- 8.....	1.020	1.017
9-11.....	1.005	8-10.....	1.020	1.018
11- 1.....	1.008	10-12.....	1.019	1.019
1- 3.....	1.005	12- 2.....	1.018	1.018
3- 5.....	1.003	2- 4.....	1.020	1.018
5- 7.....	1.005	4- 6.....	1.018	1.019
7- 9.....	1.007	6- 6.....	1.019	1.019
9-11.....	1.007			

Interpretation: Marked fixation of gravity in 1916 at a low level; in 1917 at a fairly high level; very low phthalein excretion. The functional tests indicate a severely damaged kidney in which a permanent and probably progressive nephritis has developed.

CASE 13.—I. C., aged 10 years. Chronic nephritis of three years' duration. Although the child has had albumin and casts in the urine at every examination during the last two years, her general condition is excellent, and at a glance she would be taken for an unusually healthy child. Urine: albumin, slight trace. Sediment shows a few hyaline and granular casts.

Blood urea nitrogen = 9 mg. per 100 c.c. blood. Phthalein excretion = 50 per cent.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8.....	1.021	2- 4.....	1.025
8-10.....	1.021	4- 6.....	1.025
10-12.....	1.022	6- 6.....	1.026
12- 2.....	1.026		

Night urine, 220 c.c.; nitrogen concentration, 1.4 per cent.

Interpretation: Moderate degree of fixation at a high level; night urine of high gravity and high nitrogen concentration; moderately low phthalein excretion; tests indicate moderate functional impairment. Taken in conjunction with the good clinical condition, they indicate a nephritis of mild character, probably not rapidly progressive.

CASE 14.—L. S., aged 7¼ years. Chronic nephritis of two years' duration; acute exacerbation; general condition poor; considerable anemia. Urine: albumin, slight trace. Sediment shows many red blood cells and granular casts. Phthalein excretion = 50 per cent.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8.....	1.015	2- 4.....	1.015
8-10.....	1.015	4- 6.....	1.016
10-12.	1.016	6- 6.....	1.017
12- 2.....	.....		

Night urine 350 c.c.

Interpretation: Marked fixation at a low level; large amount of night urine of a low gravity; moderately low phthalein excretion. In conjunction with the general poor condition the tests indicate a permanently damaged kidney, with an ultimately poor prognosis.

CASE 15.—H. P., aged 3 years. Chronic nephritis of one year's duration. Urine: albumin, trace. Sediment shows rare hyaline and granular cast. Blood urea nitrogen = 14 mg. per 100 c.c. blood. Phthalein excretion = 60 per cent.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	.....	2- 4	1.017
8-10	1.020	4- 6	1.018
10-12	1.020	6- 6	1.015
12- 2	1.020		

Night urine, 210 c.c.; nitrogen concentration, 0.70 per cent.

Interpretation: Fixation of gravity, during the day especially; low gravity and low nitrogen concentration of night urine; low normal phthalein excretion. We can say from the functional tests that the kidney at present is slightly damaged. Whether or not the damage will be permanent and progressive cannot at present be foretold.

CASE 16.—J. G., aged 6 years. Chronic nephritis; acute exacerbation; tests made while he was eliminating edema. Urine: albumin, large trace. Sediment shows many hyaline and granular casts. Blood urea nitrogen = 25 mg. per 100 c.c. blood. Phthalein excretion = 50 per cent.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	1.011	2- 4	1.014
8-10	1.013	4- 6	.....
10-12	1.010	6- 6	1.020
12- 2	1.014		

Interpretation: Fixation of gravity during the day. The gravity is of little significance in this case as he was eliminating edema. The test diet caused a marked diuresis; his fluid intake was 900 c.c. and his output 1,365 c.c. for the twenty-four hours. With this diuresis he eliminated 10.9 gm. of salt on an intake of about half this.

CASE 17.—F. O'L., aged 8 years. Chronic nephritis secondary to scarlet fever; two years' duration; acute exacerbation. Urine: albumin, large trace. Sediment shows numerous red blood cells and granular casts. Blood urea nitrogen = 16 mg. per 100 c.c. blood. Phthalein excretion = 59 per cent.

Time	Sp. Gr.	Time	Sp. Gr.
6- 8	.....	2- 4	1.019
8-10	1.017	4- 6	1.018
10-12	1.018	6- 6	1.016
12- 2	1.019		

Night urine 500 c.c.

Interpretation: Marked fixation of gravity with a large amount of night urine of low gravity. Phthalein excretion very slightly diminished. Blood urea normal. Two-hour renal test shows a diminution of kidney efficiency; other tests do not.

DISCUSSION OF "TWO-HOUR" RENAL TEST

The "two-hour" renal test is a test of kidney function that is particularly well suited for use in children, as it takes only a day to perform, complicated chemical analyses are not needed, and strict dieting is unnecessary. Fifteen normal cases were studied, and in none of them was there a fixation of gravity, or a night urine of low gravity; therefore, there is a good standard of normality. In nearly all the nephritis cases studied there was a definite fixation of gravity, sometimes to an extreme degree, and many cases showed a night urine of low gravity. The test is a very delicate one, probably more so than any other, and it is possible that relatively marked fixation of gravity may mean only slight functional damage. A good deal of caution is

necessary, therefore, in the interpretation of this test, and in stating an opinion on any given case it is best also to take into consideration the phthtalein test, the urinary examination, and the general clinical condition of the child.

Blood Urea Nitrogen. — It has long been recognized that end-products of protein metabolism are sometimes retained in the blood in cases of nephritis. The determination of the concentration in the blood of these products is considered to be a valuable diagnostic and prognostic aid. The concentration of urea nitrogen gives us a very good idea of the concentration of the nitrogenous end-products, and the urea nitrogen is the substance that is being used by most observers as an index of nitrogen retention.

Leopold and Bernhard,[9] studying normal children, found that there was a good deal of variation in the concentration of urea nitrogen in the blood. Their lowest figure was 8 mg. per 100 c.c. of blood, and their highest 21 mg., giving an average of 12 mg. for fifty cases. Inasmuch as only one of their normal cases was over 15 mg., and this one was a cardiac patient, who might well have had urea retention, it is fair to assume that any concentration of blood urea nitrogen over 15 mg. per 100 c.c. of blood is abnormal.

We have made blood urea nitrogen determinations on twelve nephritic children, using the urease method.

TABLE 4.—Blood Nitrogen Determination in Nephritic Children

Case	Urea Nitrogen Per 100 C.c. Blood, Mg.	Remarks
1. J. C.	32.0	Acute nephritis; no edema
2. W. K.	36.0	Acute nephritis; much edema; very scanty urine excretion
3. J. M.	11.6	Severe acute nephritis; considerable edema
4. H. K.	23.0	Severe acute nephritis; much edema
5. M. K.	46.0	Severe acute nephritis; much edema; died the next day
6. R. M.	21.0	Severe acute nephritis; much edema
7. F. O'L.	16.0	Chronic nephritis (mild)
8. M. H.	14.0	Chronic nephritis (mild)
9. H. P.	14.0	Moderately severe chronic nephritis
10. S. W.	24.0	Severe chronic nephritis; died three days later
11. J. C.	25.0	Chronic nephritis
12 J. C.	9.0	Chronic nephritis

DISCUSSION OF BLOOD UREA NITROGEN DETERMINATIONS

Of course, this series of cases is by far too small on which to base any conclusions. From a study of these few cases it would seem that in the severe cases of acute nephritis there may be considerable urea retention, and that a high blood urea warns us to be on the watch for uremia. The same information is given to us by the clinical condition

9. Leopold and Bernhard: Am. Jour. Dis. Child., 1916, **11**, 432.

of the patient. Most of the chronic cases of nephritis in children are so mild and so slowly progressive that there is no urea retention, or at least not enough to give us any information of value. From the little experience that we have had with blood urea determinations we should be inclined to conclude that they have comparatively little value in dealing with nephritis in children, and that they apply more particularly to the hypertensive cases with contracted kidneys, seen in adults.

SUMMARY AND CONCLUSIONS

Four methods of testing kidney function in children have been discussed: the added salt and urea test, the phthalein test, the two-hour renal test, and the determination of the concentration of urea nitrogen in the blood. Of these tests the added salt and urea test is of little practical value in children. The determination of the blood urea is probably of slightly more value. The most valuable of the four tests discussed are the phthalein test and the two-hour renal test, because they are simple to carry out, and because they give us reliable and important information concerning the functional power of the kidney. These two tests supplement each other, and more valuable information can be obtained by using them together than by using either one singly. I feel that these two tests are of unquestioned value, not in diagnosis, not as guides to treatment, but particularly in *prognosis*. The prognosis of nephritis in early life is most difficult, and who can say whether any given child with mild subacute or chronic nephritis will ultimately recover, or whether the nephritis will progress, with a finally fatal termination in a few years? We know that many of these patients recover entirely after one or more years of continuous or intermittent albuminuria, and again, that many of them develop a permanent chronic nephritis and die within a few years.

The tests for function, taken in conjunction with the general condition of the child, help us a good deal in giving an intelligent prognosis, but even with the tests we are often at a loss. In general, repeated *low* functional tests at intervals of a few months mean a poor prognosis. High phthalein excretion alone does not help us one way or the other. High phthalein excretion, normal blood urea, and a normal response to the two-hour test warrant a conclusion that the process is a mild one, that the kidneys are only slightly damaged, and that there is a good chance for ultimate recovery.[10] The diagnosis and treatment of nephritis in children is comparatively simple, but the prognosis is difficult and of great importance, and any simple laboratory tests that can help us in answering this question are worthy of general consideration and use.

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10. The question of prognosis will be taken up more in detail in a later paper.

THE PROPHYLACTIC USE OF PERTUSSIS VACCINE CONTROLLED BY THE COMPLEMENT FIXATION TEST *

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During the past five years numerous clinical reports have been made on the prophylactic and curative value of pertussis vaccine. Graham,[1] Ladd[2] and Bamberger[3] reported favorably on its curative power in small doses. Hess[4] was fortunate in being able to observe the effect of vaccines during an epidemic of whooping cough in an institution; he was skeptical of its curative power, but was favorably impressed by its prophylactic value, even though 10 per cent. of his treated patients developed pertussis. Hartshorn and Moeller,[5] after reviewing the literature and reporting cases of their own, thought the vaccine worthy of further trial, but did not recommend its general use. Abt[6] says that "the possibility of immunizing by vaccination is still an open question for pertussis." Luttinger,[7] after an extensive experience with the whooping-cough clinic of the New York City Health Department, concludes that pertussis vaccine in large doses (that is, one-half billion, one billion and two billion bacteria) is of value in preventing whooping cough. In his most recent report[8] he says that "his results would warrant the routine administration of pertussis vaccine for both curative and prophylactic purposes." Von Sholly, Blum and Smith,[9] using the same material as Luttinger, conclude that "more observations and more critical observations with controls for comparison must be made before the case can be made out for the curative and prophylactic value of pertussis vaccine."

It was thought that this question could be attacked from a new angle. The observations on the results of vaccines made thus far have been chiefly clinical. If vaccines are of value it would be natural to

* Submitted for publication June 11, 1917.

* From the Department of Pediatrics and the Department of Pathology and Bacteriology, University of Minnesota.

1. Graham, E. E.: Am. Jour. Dis. Child., 1912, **3,** 41.
2. Ladd: Arch. Pediat., 1912, **29,** 581.
3. Bamberger: Am. Jour. Dis. Child., 1913, **5,** 33.
4. Hess, A. F.: Jour. Am. Med. Assn., 1914, **63,** 1007.
5. Hartshorn and Moeller: Arch. Pediat., 1914, **31,** 586.
6. Abt: Arch. Pediat., 1916, **33,** 881.
7. Luttinger: New York Med. Jour., 1915, **101,** 1043.
8. Luttinger: Jour. Am. Med. Assn., 1917, **68,** 1461.
9. Von Sholly, Blum and Smith: Jour. Am. Med. Assn., 1917, **68,** 1451.

expect that after their administration specific antibodies would be present in the blood. We have two methods for determining the presence of specific antibodies — the agglutination and complement fixation tests. The agglutination test in pertussis has been tried by many workers, Shiga,[10] Wollstein,[11] Povitzsky and Worth[12] and others, and all agree that it is far from satisfactory. The complement fixation reaction, however, has been more successful. Bordet and Gengou,[13] using the bacillus discovered by them as antigen, found a positive complement fixation test in all cases of pertussis. Bächer and Menschikoff[14] found positive complement fixation in pertussis only after the injection of vaccine, but clinically noticed no benefit from the use of the vaccine. Shiga, Imai and Eguchi[10] were able to differentiate between *B. influenza* and the Bordet-Gengou bacillus by means of complement fixation. Wollstein[11] in nine cases of pertussis was unable to make the complement fixation test react positively. Olmstead and Povitzsky[15] found that "the separation by morphologic and cultural characteristics of the typical Bordet-Gengou bacillus from atypical strains of the Bordet-Gengou bacillus and from the influenza bacillus has been confirmed by complement fixation tests." Winholt[16] found a positive complement fixation obtainable two weeks, but stronger about eight to ten weeks, after the onset of the disease, and that when the influenza bacillus was used as antigen with serum of pertussis patients, no complement fixation occurs. Friedlander,[17] by a slight modification of technic, obtained positive complement fixation in all cases of pertussis even in the early catarrhal stages.

It was, therefore, proposed to select a number of healthy children who, as far as could be determined, had never had whooping cough, and vaccinate them with different pertussis vaccines, in order to study the effect of such treatment on antibody formation, by means of the complement fixation test.

TECHNIC

Two types of vaccines were employed:[18] a commercial vaccine, purchased in the open market, and a vaccine made by the ordinary method of heating the bacteria for one hour at 60 C. The latter was standardized, in the usual manner, by counting against the red blood corpuscles of normal blood. Two

10. Shiga, Imai and Eguchi: Centralbl. f. Bakt., abt. 1, 1912.
11. Wollstein: Jour. Exper. Med., 1909, **11,** 41.
12. Povitzsky and Worth: Arch. Int. Med., 1916, **17,** 279.
13. Bordet and Gengou: Ann. d. l'Inst. Pasteur, 1906, **20,** 218.
14. Bächer and Menschikoff: Centralbl. f. Bakt., abt. 1, 1912, **61,** 218.
15. Olmstead and Povitzsky: Jour. Med. Research, 1916, **33,** 379.
16. Winholt: Jour. Infect. Dis., 1915, **16,** 397.
17. Friedlander and Wagner: Am. Jour. Dis. Child., 1915, **8,** 135.
18. I am indebted to Dr. W. P. Larson for valuable assistance with the laboratory work.

different strains of Bordet-Gengou bacillus were used, one obtained from the New York City Department of Health and the other from a commercial biological house.

The technic of the complement fixation tests was that ordinarily used. About 1 c.c. of the patient's blood was drawn and allowed to coagulate; the serum was separated in the centrifuge and heated to 56 C. for ten minutes (we find that ten minutes is sufficient to inactivate the serum and there is less danger of impairing its antibody content); in the tests 1 to 2 drops of undiluted serum from a capillary pipet (of which about 20 drops make 1 c.c.) was taken. The amboceptor consisted of antisheep rabbit serum (1:40). The antigen was prepared by scraping, in distilled water, a forty-eight hour growth of *Bacillus pertussis* on blood agar and shaking for twelve hours, after which it was incubated at 48 C. for twenty-four hours. The antigen was standardized against the blood serum of a known case of pertussis.

The attempt was made to immunize rabbits by injecting prophylactic doses of vaccines and, at the same time, inoculating control rabbits with living Bordet-Gengou bacilli. A comparison of the antibody production of living and dead bacilli was rendered impossible by the fact that many rabbits gave a positive complement fixation test before treatment was begun. The tests had, therefore, to be made entirely on humans. For this purpose fairly normal and healthy children, ranging from 6 months to 3 years, were chosen from the inmates of Lymanhurst, City Children's Hospital.

In all, seventeen patients were treated; these were divided into three series. In the first series of four cases, three were given vaccine prepared by us and one a commercial vaccine, once a week for four weeks; the first two doses were one hundred million bacilli, and the last two doses two hundred million. Blood for the complement fixation test was taken before each injection, and again two weeks after the last injection. All tests were absolutely negative. In the second series of four cases, all were given our vaccine, two hundred million being given once a week for five weeks. Complement fixation tests performed before each injection and one performed two weeks after the last injection were, likewise, all negative. In the third and last series, the dosage recommended by the New York City Health Department was used; one half billion, one billion and two billion bacteria were given at two-day intervals. There were nine cases; of these, five received our vaccine and four received commercial vaccine. Of the five who received freshly prepared vaccine, three reacted positively and two were negative. Of the four cases receiving commercial vaccine, one reacted positively and three negatively. In this last series of nine cases the ages ranged from 6 months to 3 years. The ages of those giving positive reaction were 12, 24, 26 and 36 months, respectively; of those giving negative reactions, 6, 14, 14½, 16 and 20 months.

SUMMARY AND CONCLUSIONS

Our observations in seventeen cases would theoretically justify the prophylactic vaccination against pertussis.

In none of the cases treated with the smaller doses could any antibodies be demonstrated. Whereas, large doses, such as recommended by the New York City Health Department, showed antibodies in 44 per cent. of the cases. The freshly prepared vaccines were, apparently, more effective than the stock vaccines, as 60 per cent. of the patients treated gave positive fixation reaction. From this, it may be concluded that it is possible to immunize children against pertussis if sufficiently large doses of freshly prepared vaccine be used.

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PAROXYSMAL TACHYCARDIA IN CHILDHOOD*

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The function of originating and transmitting the automatic impulses which result in the contraction of the heart belongs to certain specialized tissues which represent the remnant of the primitive cardiac tube. These excitation waves have been shown to arise at the junction of the superior vena cava with the right auricle, in the upper portion of the sino-auricular node. From here they pass through the node and are transmitted by numerous paths to the auricular musculature and to the atrioventricular node described by Tawara. From this node stimuli pass through the bundle of His and into its branches, which divide and subdivide until their ramifications form an intricate network by means of which the impulses are rapidly distributed to the ventricular myocardium. As impulses travel downward through these conducting fibers, the heart responds by sequential contractions of the auricles and ventricles. Under certain conditions, however, any portion of this conducting system may acquire the function of originating the excitation wave, producing variations in rate and rhythm and occasionally a complete reversal of the cardiac mechanism.

Vagal influences or a local lesion may so depress the normal point of origin that some other locality may assume the rôle of pacemaker. A lesion of the auricular or ventricular myocardium or endocardium may increase the sensitivity of some portion of this specialized tissue and likewise establish an abnormal rhythm. Lesions may directly involve any of the conducting fibers, most frequently those at the atrioventricular node, and produce a delay in transmission or an actual blocking of the excitation wave, which in some instances reaches its destination by an abnormal and more circuitous route. The conductivity of these tissues may also be so reduced by the transmission of impulses arriving with excessive frequency that a functional delay or even a fatigue block may result. All of these conditions affect the electrocardiographic picture and the characteristic changes are now well recognized.

In auricular flutter and in paroxysmal tachycardia the heart contracts in response to excitation waves of ectopic origin which arise below the sino-auricular node at a rate of 200, or more, a minute. In both conditions the auricle responds to each stimulus, but the ven-

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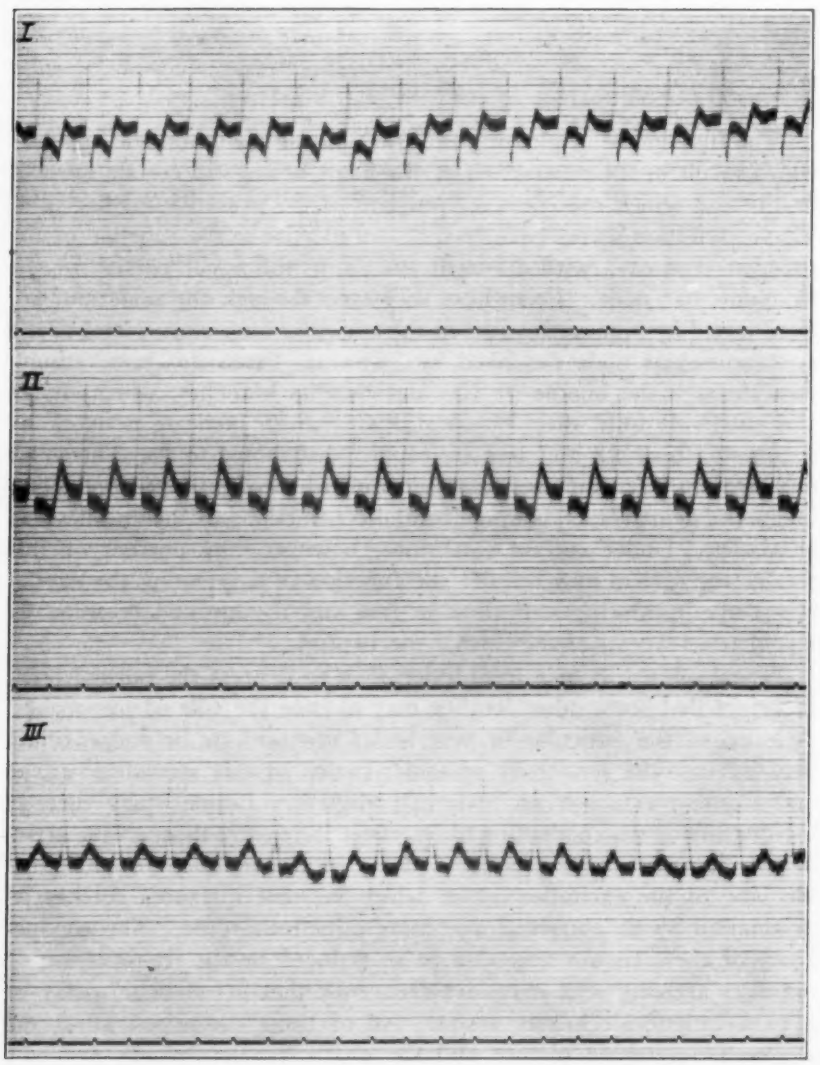

Fig. 1.—Electrocardiogram taken during paroxysm. Derivations I, II and III. Rate, 196.5 per minute. The P wave is represented by a deflection between S and T. Standardized; 1 cm. deflection = 1 millivolt. Time recorded in fifths of a second.

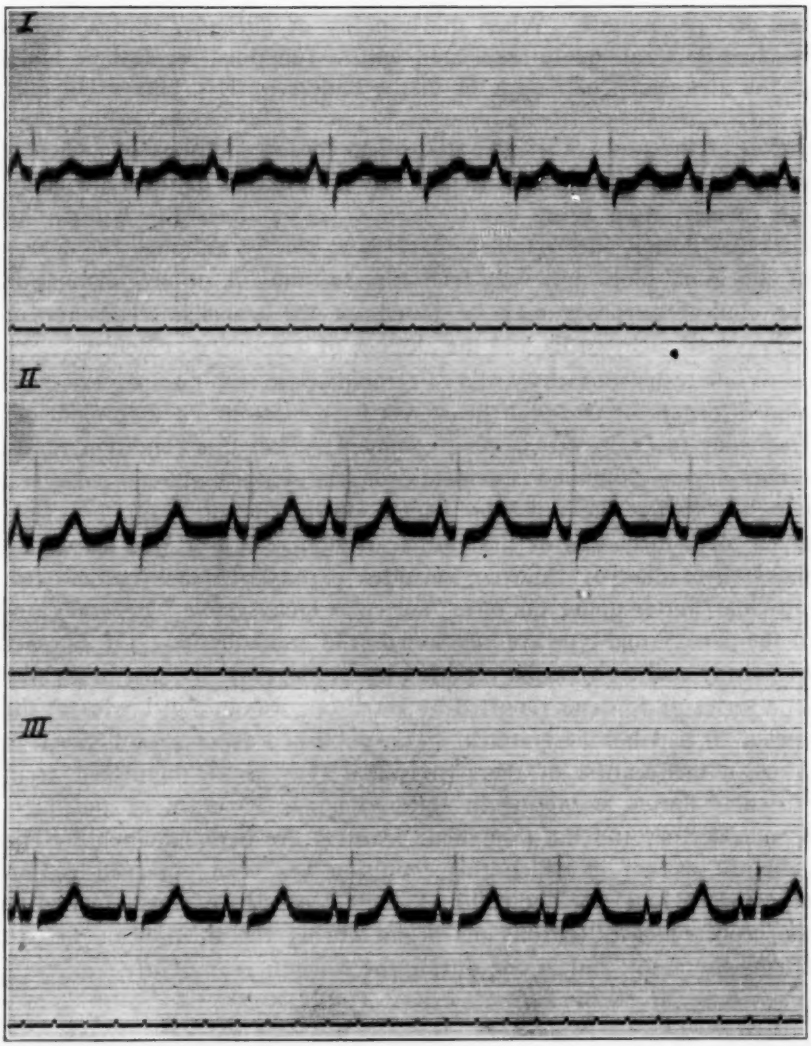

Fig. 2.—Electrocardiogram taken between paroxysms. Derivations I, II and III. Rate varies from 96 to 103 per minute. Standardized; 1 cm. deflection = 1 millivolt. Time recorded in fifths of a second.

tricular response is largely dependent on the ability of the conducting fibers to transmit excitation waves at this rapid rate. In paroxysmal tachycardia the auricle seldom acquires a rate of more than 200 in adults or of 250 in children. In flutter the rate is usually between 240 and 360 per minute. When this rapidity of auricular contraction is reached it is commonly associated with a functional or fatigue block at the atrioventricular node or in the bundle of His, permitting only a certain proportion of the impulses to pass. This has led to the application of the term "auricular flutter" to those conditions of auricular tachysystole in which the auricular rate is in excess of the ventricular, the former often being exactly two or more times the latter. An auricular flutter, once established, tends to persist, although the degree of functional block may vary from time to time. A suitable distinction for clinical purposes may be based on the transient character of paroxysmal tachycardia and the absence of a ventricular deficit.

While transient tachycardia of ectopic origin is not among the common disorders of the adult heart, yet it is encountered with sufficient frequency to excite but passing comment. That this condition rarely occurs during childhood is evident from the scarcity of reported cases, notwithstanding the attention which, during recent years, has been given to disturbances of the cardiac rhythm. Wilson,[1] in discussing the tachycardias of children, includes paroxysmal tachycardia with auricular flutter under the term "auricular tachysystole." Several cases of the paroxysmal type are mentioned in his contribution. Of 29 cases of paroxysmal tachycardia reported by Lewis, only one was in a child. In a series of 33 cases reported by Hart[2] one was in a girl, who, at the age of 9, had her first attack. The published reports of 9 cases in which a tachycardia of the paroxysmal type, as distinguished from auricular flutter, occurring in children under 10 years of age, have been collected, and the prominent features are here tabulated.[3] The youngest patient in which this condition has been observed is that reported by Lewis.[4] The child was only 3 months of age. In two of the reported cases, paroxysms began during the third year, in two during the sixth, and in one during the seventh year of life.

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1. Wilson, F. N.: Recent Progress in Pediatrics, Am. Jour. Dis. Child., 1915, **10,** 376.

2. Hart, T. S.: Abnormal Myocardial Function, 1917, p. 95.

3. In addition to the cases in the table, there are in the records of the Mt. Sinai Hospital electrocardiograms of several children which show typical paroxysmal tachycardia. These were included in the electrocardiographic exhibit of that institution at the Annual Meeting of the American Medical Association, New York, 1917, but are still unpublished.

4. Lewis, T.: Lectures on the Heart, 1915, p. 117. (Delivered at McGill University, 1914.)

The occurrence of paroxysmal tachycardia in young children has been brought to our attention by the case of a patient recently admitted to the Harriet Lane Hospital in the service of Dr. John Howland, who has kindly furnished the clinical data for the following report.

REPORT OF CASE

M. P. L., a sturdy boy 3½ years of age, was admitted May 12, 1917, to the Harriet Lane Home of the Johns Hopkins Hospital, suffering from frequent attacks of tachycardia. He is the younger of two children, his brother being in perfect health. His parents are living and well. At birth the patient weighed 7¼ pounds. When 3 months of age he weighed 12 pounds. At this time artificial feeding was begun and various milk modifications were employed. Nutritional disturbances were constant during the year and a half following. At 12 months the child weighed only 11½ pounds. During this period he had frequent attacks of vomiting and it was noticed that at these times the heart's action was extremely rapid. The vomiting was so persistent that a diagnosis of probable pyloric stenosis had been made.

During his second year he was placed on a more liberal diet, and his condition began to improve. Since then he has developed normally and remained in excellent health. During the past year he has vomited three times, but only once on each occasion. The attacks of rapid heart action have continued, coming usually every three or four days, sometimes more often. Occasionally several weeks have elapsed between attacks. The parents become aware of the presence of the tachycardia by a pallor of the child's face, slight engorgement of the veins of his forehead and by the excessive and rapid pulsation in his neck. He becomes somewhat fretful and less active, but usually continues his play and suffers no apparent inconvenience. The attacks last from two to thirty-six hours, usually three or four; begin abruptly without premonition and stop suddenly. It has been thought that excitement and excessive activity might be contributing factors. On the contrary, the tachycardia has occasionally developed while the child was asleep.

He was examined May 12 by Dr. Howland, who reports:

"He is a well nourished, alert, active child. The posterior cervical glands on the left side are moderately enlarged. There is no other glandular enlargement. The tongue is clean. The apex is in the fourth interspace, in the nipple line. There is a slight sinus arrhythmia. There is a cardiorespiratory murmur heard best in the second right interspace, close to the sternum, heard only during midexpiration. It is heard also in the interscapular region behind. The lungs are normal; spleen not palpable; von Pirquet reaction negative."

May 14 the child experienced a typical attack. During the morning while playing out of doors a slight pallor of his face was noticed and a rapid, forcible pulsation of the jugular vein was observed. He was entirely free from discomfort, and continued bright and playful. The pulse was about 200. After two hours the attack subsided and the pulse dropped to 112. The patient was discharged May 15. Reports indicate that the paroxysms have since then been of less frequent occurrence. There were no attacks during the first three weeks in June.

Electrocardiographic records were obtained May 13, 14 and 15. One of these was taken during the paroxysm which occurred on the 14th. The rate computed from the electrocardiogram is 196.5 per minute. The sequence is normal. Following an R wave of supraventricular origin, there is, in the first and second derivations, a deep S, interrupted in its ascent by an inverted P wave, which, with the ascending limb of the following T, results in a large diphasic curve. In the third derivation the P wave is represented by a small oscillation between the R and T waves. Conductivity is diminished, the P-R interval measuring 0.21 second. The electrocardiogram is typical of a paroxysmal tachycardia of auricular origin (Fig. 1).

REPORTED CASES OF PAROXYSMAL—

Reported by	Date	Age	Sex	Predisposing Factors	Age When Paroxysms Began, Yrs.	Number of Paroxysms	Duration of Paroxysms	Normal Pulse
Buckland,[5] F. O.	1892	11	♀	Measles; neurotic temperament	5	Numerous; two attacks observed	12 hours to 10 days	84 to 96
Herringham,[6] W. E.	1898	11	♀	Scarlet fever during infancy; tonsillitis; constipation	5	Seven in 10 months	18 hours to 13 days	80 to 96
Merklen,[7] P.	1901	13	♀		Not stated	Occasional	12 hours to 8 days	80
Hay,[8] John	1907	6	♂	"Delicate constitution"	6	Frequent; almost continuous for six months	Few seconds to 10 days	84
Hume,[9] Wm. E.	1913	6	♀	Occurred during convalescence from diphtheria after antitoxin treatment	6	Numerous on 41st and 51st days of illness	"Short and long periods"	88
Kidd,[10] Percy	1914	4½	♂	Measles	2	About once each month; frequent during last six months	Few minutes; later almost continuous	80
Hutchinson,[11] R., and Parkinson, J.	1914	2¾	♂	No previous illness; well nourished	2¼	Four in five months	12 hours to 12 days	64 to 80
Lewis,[4] Thomas	1914	3 mos.	..		3 mos.		Several hours	
Hart,[2] T. S.	1917	10	♀		9			
Personal observation	1917	3½	♂	Gastrointestinal disorders and malnutrition during infancy	During 1st yr.	Usually every 3 or 4 days	2 to 36 hours	90 to 112

Electrocardiograms taken on the 13th and 15th have a normal rhythm and a normal sequence. That taken on the 15th shows a rate which varies from 96 to 103 per minute. There is a moderate sinus arrhythmia. The P wave in the first derivation is slightly notched. The S deflection is deep in the first and second derivations. Conductivity is normal, the P-R interval measuring 0.14 second. The electrocardiogram is normal (Fig. 2).

5. Buckland, F. O.: Case of Rapid Heart, Tr. Clin. Soc. Lond., 1892, **25,** 92.

6. Herringham, W. P.: A Case of Paroxysmal Tachycardia, Tr. Clin. Soc. Lond., 1898, **30,** 99.

7. Merklen, T.: Tachycardie paroxystique essentielle chez l'enfante, Semaine méd., 1901, **31,** 164.

8. Hay, John: Paroxysmal Tachycardia (Case 2), Edin. Med. Jour., 1907, **21,** 40.

9. Hume, W. E.: Irregularities in Diphtheria (Case 4), Heart, London, 1913, **5,** 25.

10. Kidd, Percy: Paroxysmal Tachycardia in a Boy Aged Four and a Half Years, Brit. Jour. Child. Dis., 1914, **11,** 264.

11. Hutchinson, R., and Parkinson, J.: Paroxysmal Tachycardia in a Child Aged Two and Three-Fourths Years, Brit. Jour. Child. Dis., 1914, **11,** 241.

—TACHYCARDIA IN CHILDREN

Pulse During Paroxysm	Paroxysm Begins	Paroxysm Ends	Accompanied by	Subsequent Course	Graphic Records
200 to 215	Abruptly, with arrhythmia	During sleep	Pallor; dizziness; rapid breathing; low fever	Probable recovery	None
200 to 256	Abruptly; often with sudden movement	Always during sleep	Languor; slight cyanosis; cardiac dilatation; enlargement of liver; jugular pulsation	Recovery	None
220	Abruptly	Abruptly; often with coughing and hemoptysis	Dyspnea; cardiac dilatation; epigastric hyperesthesia; hemoptysis	Unknown	None
182 to 230	Abruptly; sometimes with vomiting	Abruptly; often with periods of arrhythmia	Pallor; listlessness; pulmonary edema; cardiac dilatation; hepatic enlargement; jugular pulsation	Recovery	Polygram; ventricular venous pulse during paroxysms; numerous auricular extrasystoles as attacks subside
225	.....	.....	Collapse; nausea and vomiting; cardiac dilatation	Recovery	Polygram; tachycardia apparently of nodal origin; extrasystoles precede paroxysm
240	Abruptly; with momentary collapse	Abruptly; sometimes after vomiting	Cardiac dilatation; hepatic enlargement; general anasarca; dyspnea	Fatal termination; probably from malignant endocarditis	None
160 to 245	Abruptly; sometimes with momentary collapse	Abruptly; once after defecation; often with arrhythmia	Vomiting; cardiac dilatation; slight edema; enlarged and pulsating liver; partial suppression of urine; jugular pulsation	Recovery	Polygram; ventricular venous pulse during paroxysms; electrocardiogram; R modified by inverted P; nodal origin (?)
270 to 290	.....	.....	.....	.....	Electrocardiogram; inverted P between R and T; probably auricular tachycardia
.....	.....	.....	.....	.....	Electrocardiogram
180 to 200	Abruptly; no premonition	Abruptly; with sense of relief; often during sleep	Slight pallor; lessened activity; jugular pulsation	Reduction in frequency and severity of paroxysms	Electrocardiogram; inverted P with beginning of T wave; auricular tachycardia

From a theoretical standpoint the excitation waves of paroxysmal tachycardia may originate in the lower portion of the sino-auricular node, in the wall of the auricle, in the atrioventricular node, in the bundle of His and its branches, or in the ventricle. Electrocardiographically, three types are recognized: auricular, nodal (atrioventricular) and ventricular. In this instance the form of the ventricular complex, Q-R-S-T, eliminates a tachycardia originating below the node of Tawara. Were the impulses originating in the atrioventricular node, the P wave would be more likely to fall with, or nearer to, the wave of ventricular excitation. In this case the P deflection is removed from the preceding R by a distance equivalent in time to 0.11 second. The presence of an inverted P indicates an impulse of ectopic origin arising in the auricle probably near the atrioventricular node. The prolonged conduction time, as measured from the inverted P to the following R, results from bundle fatigue due to the very frequent stimuli which this

structure is called on to transmit. The consistent form of the atypical P waves suggests that the stimuli come from a single focus. It is reasonable to conceive of this tachycardia as a rapid succession of extrasystoles, originating at some point in the auricle which, during the paroxysm, assumes the rôle of pacemaker.

The etiology of paroxysmal tachycardia is obscure. Alcohol, tobacco, gastro-intestinal disturbances, inflammation of the upper respiratory tract, infectious diseases, arthritis, endocarditis, nephritis, hypertension and coronary sclerosis have been mentioned as predisposing causes. In the series collected of paroxysmal tachycardia in children under 10, diphtheria, measles and scarlet fever are given as predisposing factors. One is described as having a "delicate constitution," while another is "well nourished, with no previous illness." The case here described has suffered from none of the infectious diseases of childhood, and the only possible predisposing condition was the early malnutrition resulting from digestive disturbances. Since the paroxysms continue and the child is in perfect health, it is necessary to assume the existence of some local lesion or defect. Whether this is a temporary depression of the sinus, such as has been produced experimentally in animals, with an escape of the auricle, or whether there is a point of hypersensitivity in the auricle with a high rate of stimulus production, it is not possible to determine. Vomiting is frequently associated with these tachycardias. This may point to slight digestive derangements, perhaps unrecognized, but sufficient to incite the paroxysm. On the other hand, the persistence of attacks over considerable periods of time during which the patients are in perfect health discounts such a supposition.

The paroxysms are usually accompanied by more or less circulatory embarrassment, and in prolonged attacks by cardiac dilatation and other signs of myocardial exhaustion. The prognosis is dependent on the ability of the heart to maintain its function during protracted paroxysms. Of the ten tabulated cases, five are reported as having recovered, while only one led to a fatal termination, and this was probably due to a malignant endocarditis which complicated the tachycardia. In young children without other cardiac disorders the prognosis is evidently favorable, and with the observation of reasonable precautions the paroxysms tend to become shorter, to occur less frequently and ultimately to disappear.

No consistent results have been obtained from medication. Various remedies have been employed only to be discarded. In the series tabulated it is interesting to note that vomiting, coughing and defecation sometimes accompany the cessation of the paroxysm, suggesting the influence of reflex inhibitory stimulation. Pressure on the vagus, ocular pressure and holding the breath have occasionally, in adults,

been followed by a termination of the attack, but usually none of these procedures is successful. Digitalis is useful when evidences of myocardial insufficiency are present, but its administration has no apparent effect on the tachycardia. All possible sources of local irritation or infection should be removed. Rest and quiet during the paroxysm, a suitable diet, proper elimination, and a life free from unusual excitement, constitute the the treatment of uncomplicated cases.

CLINICAL DEPARTMENT

A CASE OF PNEUMONIA OF UNUSUALLY SHORT DURATION *

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Abortive pneumonia is the term applied by Wunderlich[1] to that class of cases presenting the clinical picture of pneumonia together with the usual signs of pulmonary consolidation, yet running an exceedingly brief and mild course, often terminating by crisis on the second or third day. Credit is given Eugen Seitz by Krafft[2] for first describing this condition. Following him it has been described under various names, pneumonic febricula (Bernheim), peripneumonic synocha (Marrotte), hemopneumonia (Woillez), and rudimentary pneumonia (Finkler). The later reports of Bechtold[3] in 1905, and of Krafft[2] and Kerr[4] in 1910, have made the condition more or less familiar.

The statement of Aufrecht[5] that the so-called abortive pneumonia is far more frequent than the reported cases would indicate, is probably correct. Because of the brevity of the disease, many cases are not seen by the physician and the diagnosis in any event is a difficult one except with the aid of accessory diagnostic measures.

The clinical picture has been analyzed in the reports mentioned. Except in duration, it does not differ materially from that of pneumonia running the usual course. The onset is usually sudden, with prostration, high fever, rapid respiration, pain in the side and cough. Typical "rusty" sputum is frequent with older patients. Kühn[6] says that herpes is more common in this than in the usual type of disease.

The presence and nature of the physical signs are variable. In the majority of cases reported there is a high pitched or dull percussion note, with crackling râles and suppressed breathing over a limited area. These signs may be present from the onset, but more frequently come out from twelve to fourteen hours later. The changes usually persist for from one to five days after the temperature becomes normal.

* Submitted for publication Aug. 5, 1917.
* From the Harriet Lane Home of the Johns Hopkins Hospital.

1. Wunderlich: Specielle Pathologie und Therapie, Stuttgart, 1856, **3,** 2.
2. Krafft: Dissertation, Jena, 1910.
3. Bechtold, A.: München. med. Wchnschr., 1905, **52,** 2113.
4. Kerr: Med. Rec., New York, 1910, **77,** 701.
5. Aufrecht, E.: Spec. Path. u. Therap., Nothnagel. 1899, **14,** 128.
6. Kühn, See G.: Diseases of the Lungs, 1891, **2,** 180.

In two of Bechtold's cases, one of Krafft's, and one of Kerr's, there were no physical signs to be elicited.

The bacteriologic evidence of abortive pneumonia is exceedingly meager. In one of Krafft's cases the pneumococcus was found in the blood; in two it was in the sputum. The organism has never been grouped.

As to the pathology of these cases, there are no available data, since there is no mortality. There has been much speculation as to whether the process passes through the stages of congestion and hepatization as in the usual pneumonia or is arrested sooner. The opinion is generally held that the process does not pass beyond the stage of congestion. Many authors, however, like Bechtold,[3] have preferred to leave the question open, or like Norris,[7] have been content to state that in such cases the consolidation is usually not complete.

The Roentgen ray affords some help in solving this problem. It is assumed by many roentgenologists that it is only when the inflammatory process in the lungs has progressed to the stage of cellular exudation that a shadow is cast.[8] In this regard the following case is of interest.

REPORT OF CASES

CASE 1.—D. B., aged 4 years. Onset March 20, with pain in the left side, slight cough, rapid respiration and fever. He continued feverish and drowsy with rapid respiration. Examination on the fourth day showed a fairly well nourished boy, somewhat drowsy, breathing rapidly and with an occasional slight cough. There was marked herpes on the upper lip and about the left nostril. Temperature, 103 F.; respirations, 44; pulse, 120; leukocytes, 33,000. There were no changes detected on auscultation and percussion. On the following day there were distinct dulness on percussion and crepitant râles over the lower left back. On the sixth day the roentgenogram showed nothing suggestive of pneumonic consolidation. The signs and symptoms of pneumonia and the leukocytosis persisted. On the ninth day the roentgenogram showed the shadow of pneumonic consolidation of the left lower lobe. The disease terminated by crisis on the eleventh day and recovery was uneventful.

This case of frank lobar pneumonia gave clear physical signs of consolidation on the fifth day and yet there was no shadow cast until the ninth day. Shall it be assumed from this that an intense congestion may give the signs of consolidation and that a cellular exudate may not occur until the seventh or eighth day of the disease? Or shall it be assumed that a cellular exudate was present earlier but was not sufficiently dense to cast a shadow? At what point of the inflammatory reaction and by what physical condition the Roentgen-ray shadow is cast is still an open question. It is difficult to use this evidence, therefore, in the interpretation of the pathology of abortive pneumonia.

7. Norris, G. W.: Modern Med., Osler and McCrae, Ed. 2, Phila., 1913, **2**, 237.
8. Baetjer, F. H.: Personal communication, 1917.

Certainly the following case strongly suggests that the process may in some cases at least go on to the stage of hepatization.

CASE 2.—C. D., aged 3 years, was admitted to Harriet Lane Home, Dec. 16, 1916.

Past History.—Measles at 15 months; pneumonia on the right side lasting ten days and complicated by empyema. Following operation he made an uneventful recovery. November 1, six weeks before the present admission, the child was taken suddenly ill with headache, vomiting, pain in the right side and cough. He was admitted to the hospital with a temperature of 103.2 F.; leukocytes 34,500 and frank signs of consolidation of the right middle lobe. The blood culture was negative. The disease ran the usual course of pneumonia and terminated by crisis on the seventh day. Recovery was uneventful. The roentgenogram taken just before his discharge showed a slight diffuse shadow in the right side of the thorax low down, which had probably been present since the empyema, sixteen months previous.

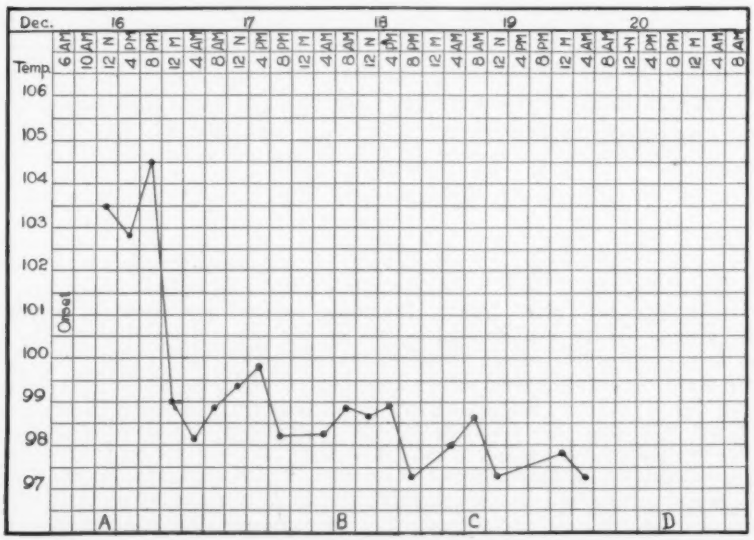

Fig. 1.—Temperature chart of a case of pneumonia, showing crisis eighteen hours after onset. *A*, white blood cells, 38,900; *B*, first roentgenogram; *C*, white blood cells, 11,000; *D*, second roentgenogram.

Present Illness.—The child was taken suddenly at 6 a. m., December 16, with headache, vomiting, high fever and rapid respiration.

When examined at 10 a. m. his cheeks were flushed. The temperature was 103.6 F., the pulse 144, the respirations 44 and the leukocytes 38,900. The chest was well formed and moved normally. There was the scar of the empyema operation below the angle of the right scapula. The dull percussion note and distant breath sounds heard over the right lower back were attributed to the thickened pleura. There was no other change detected on auscultation and percussion. The diagnosis of pneumonia was made on the history of sudden onset, the prostration and rapid breathing and the leukocytosis.

The examination at 8 p. m. showed the condition unchanged; temperature 104.5 F.; pulse, 144; respirations, 44. Defervescence occurred by crisis and at

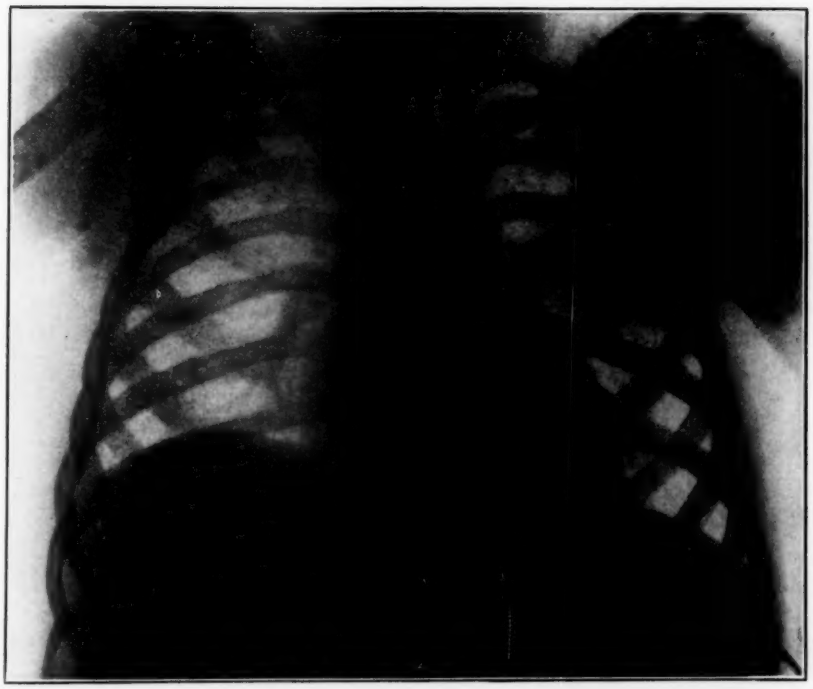

Fig. 2.—Roentgenogram showing triangular area of consolidation in the upper part of the left lung.

Fig. 3.—Roentgenogram showing resolving consolidation at the upper part of left lung.

12 midnight; eighteen hours after the onset his respiration was quiet and easy, his temperature 99 F., and his pulse 100.

The temperature did not rise above 100 F. subsequently. The following day the patient was sitting up, bright and apparently free from discomfort. The roentgenogram (Fig. 2) showed a triangular area of consolidation peripherally located in the upper part of the left lung.

December 19, leukocytosis, 11,000. Recovery was complete.

Roentgenogram December 21 (Fig. 3), four days after the crisis, showed very slight remains of the consolidation.

The history of the above case is typical of pneumonia save in its duration and the absence of physical signs. The absence of the latter may be understood when the location of the lesion is considered. As shown by Mason,[9] consolidation peripherally located may give no changes that can be detected by auscultation. An impaired percussion note over the affected area was probably present, but was obscured by the absence of a normal note on the other side for comparison.

I have not been able to find in the literature another case of the so-called abortive pneumonia of which roentgenograms were made.[10]

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9. Mason, H. H.: Am. Jour. Dis. Child., 1916, **11,** 188.

10. Other case reports: Weil, A.: Ein Fall von eintägiger Pneumonie, Berl. klin Wchnschr., 1879, **16,** 665. Simon: Zur Kasuistik der abortiven Pneumonie, München. med. Wchnschr., 1908, **55,** 1837. Engels: Zur Kasuistik ephemer. Pneumonien, München. med. Wchnschr., 1908, **55,** 2088. Pollatschek: Zur Kasuistik der abortiven Pneumonie, München. med. Wchnschr., 1908, **55,** 2089. Ruhl: Zur Kasuistik der abortiven Pneumonie, München. med. Wchnschr., 1908, **55,** 2391.

CASE OF PERNICIOUS ANEMIA IN A BOY OF EIGHT YEARS *

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History.—Martin A. was born in August, 1908, in a small settlement in the woods of northeastern Maine and had always lived there. His parents were healthy. Four other children were alive and well; two had died in infancy of indigestion. There had been no miscarriages.

Martin was born at full term, after a normal labor. He was nursed for eighteen months and then given a general diet. He had measles when he was a year old and whooping cough when he was 4 years old. Otherwise he had always been well.

When he was 5 years old, he began to complain of being tired. He did not look well and became very pale, the skin taking on a yellowish tinge. He did not feel well enough to go to school, but was not confined to bed. He improved somewhat after six months, but soon relapsed. Since then his general condition had, on the whole, deteriorated. He was treated for a short time in the Eastern Maine General Hospital, at Bangor, when 6½ years old. The examination of his blood made there at that time was as follows:

Hemoglobin (Dare)	28 per cent.
Red blood corpuscles	1,600,000
White blood corpuscles	8,000

Both fresh and stained smears of the blood showed great variation in the size, shape and color of the red cells. About one-half of the cells were in the form of rings. They were somewhat stippled, but very few nucleated red cells were seen. A differential count showed:

	Per Cent.
Small lymphocytes	35.0
Large lymphocytes	4.0
Large mononuclears	3.66
Transitionals	1.0
Polymorphonuclear neutrophils	51.6
Polymorphonuclear basophils	0.66
Normoblasts	2.33
Unclassified	1.66

The stool at that time was formed and light-brown in color. It showed no fermentation and no microscopic blood, but contained several large food particles. No ova or parasites were seen. The Wassermann test made there was negative.

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*From the Medical and Pathological Services of the Children's Hospital.

When he was 7 years old, he was sick in bed for five weeks with a high fever and difficulty in breathing. His parents did not know what was the trouble. During the last six months he had been able to be up and about the house, but had had a poor appetite. His bowels had moved normally and the stools were thought to have been normal. He was admitted to the Children's Hospital Nov. 9, 1916, when 8 years and 3 months old.

Physical Examination.—He was fairly developed and poorly nourished. He was very pale, and there was a slight yellow tinge to the pallor. The superficial veins of the scalp were dilated and there was some edema of the back of the head. The nose and ears were normal. The tongue was pale and clean. His teeth were much decayed and there was an abscess about the left inferior six-year molar. The gums were fairly healthy. The tonsils were of moderate size and apparently healthy. The lungs showed nothing abnormal. The area of cardiac dulness extended 3 cm. to the right and 9 cm. to the left of the median line. There was a sharp systolic murmur at the aortic area, with a slight diastolic murmur down the middle of the sternum. There was a soft systolic murmur at the apex and a sharp, high-pitched diastolic murmur at the apex. The second pulmonic sound was louder than the second aortic sound. There was both a capillary and a Corrigan pulse. There was a pistolshot and marked double murmur in the groin. The systolic blood pressure was 118 and the diastolic between 45 and 50. There was a marked systolic blowing sound in the vessels of the neck. D'Espine's sign was absent. The abdomen was soft and tympanitic; there were no evidences of fluid or of new growths. The lower border of the liver was felt 5 cm. below the costal border in the nipple line. The spleen was not palpable. The genitals were normal. The extremities were normal. There was no spasm or paralysis. The knee jerks were equal and normal. Kernig's and Babinski's signs were absent. There were a number of lymph nodes in the neck, the size of marbles and almonds, those on the left being somewhat larger than those on the right. There was no enlargement of the other peripheral lymph nodes. The skin was dry and scaly. It was fairly dark-brown over the abdomen and in the axillae. There were one or two brownish patches on the legs. The scrotum was also somewhat pigmented. The mouth, however, showed no discolorations. The examinaton of the fundi showed nothing abnormal, except extreme pallor.

The examination of the blood at entrance was as follows:

Hemoglobin (Sahli)	15 per cent.
Red blood corpuscles	860,000
White blood corpuscles	8,000
Small mononuclears	61.0 per cent.
Large mononuclears	11.0 per cent.
Polynuclear neutrophils	25.0 per cent.
Eosinophils	0.5 per cent.
Basophils	2.5 per cent.

The red corpuscles showed little achromia, but marked variations in size and shape. There was a tendency to a predominance of large forms. There was an occasional stippled red cell. The platelets numbered 12,900. Ten normoblasts and one megaloblast were seen to each 200 white cells.

The urine was clear, acid, of a specific gravity of 1.011 and contained the slightest possible trace of albumin, but no sugar, acetone or bile. The sediment showed a few epithelial cells. It contained no casts or urobilinogin.

The stool was soft, acid in reaction, golden-brown in color, sour in odor. It contained no neutral fat or fatty acids, but a little soap. It showed a considerable amount of starch. No parasites or ova were seen. There was apparently a slight increase of urobilinogin in the stool.

The Wassermann and tuberculin tests were negative. Blood cultures were negative.

During the patient's stay in the hospital his temperature was usually between

100 F. and 101 F., sometimes a little lower, sometimes a little higher. His decayed teeth were extracted and the jaws and gums made healthy, without any improvement in his condition. He had no diarrhea. Repeated examinations of the stools showed constantly a slight amount of soap, but starch was never found again. An examination for occult blood, when he had been on a hemoglobin-free diet for a number of days, showed none. Parasites and ova were sought for carefully many times, but were never found. The urine never contained albumin again. The blood showed no improvement. The examination of the blood November 28, the day before his death, was as follows:

Hemoglobin (Sahli)	10 per cent.
Red corpuscles	570,000
White corpuscles	6,200

Differential count:

Small mononuclears	47 per cent.
Polymorphonuclear neutrophils	53 per cent.

The red cells showed marked variation in size and shape. Six normoblasts, one stippled cell, three polychromatophilic cells and two or three degenerated cells were seen in counting 200 white cells.

Treatment.—He received no specific treatment except one intravenous injection of 20 c.c. of whole blood with 4 c.c. of 10 per cent. solution of sodium citrate. It had been intended to try repeated injections of small amounts of blood, but his death prevented this method of treatment from being carried out.

His general condition steadily deteriorated from the time of his entrance to his death, November 29.

The diagnosis of pernicious anemia seemed very positive in this instance, although no cause for it could be made out. It hardly seemed possible that the decayed teeth and alveolar abscess could be the cause of his trouble, for while secondary anemia with leukocytosis is not uncommon as the result of decayed teeth and diseased jaws and gums, the toxemia from them would hardly be sufficient to cause so severe a condition as pernicious anemia. Addison's disease was suggested to a certain extent by the discoloration of the skin. The absence of profound muscular weakness, the normal systolic pressure and the slightly elevated temperature showed, however, that there was no adrenal insufficiency. The pigmentation could, moreover, be easily explained as the result of blood destruction. It was felt that the enlargement of the heart, the murmurs in the heart and neck, the high pulse pressure and other evidences of aortic insufficiency were not due to organic disease but to muscular weakness and vasomotor insufficiency.

The necropsy was performed December 2, by Dr. S. Burt Wohlbach and Dr. S. B. Marlow. Dr. Wohlbach's report is as follows:

Necropsy.—At the Children's Hospital, Dec. 2, 1916, on Martin A., aged 8 years.

Anatomic Diagnoses.—Anemia; emaciation; fatty degeneration of heart, liver and kidney; hypertrophy and dilatation of heart; edema of lungs, pericardium and scalp; hyperplasia of bone marrow, accompanied by marked thickening of calvarium. *Ascaris lumbricoides.*

Microscopic Diagnoses.—Acute bronchitis; acute parenchymatous degeneration of kidneys.

Body: The body is that of a well developed, emaciated white boy. The skin of the whole body is uniformly olive-brown in color. There is deeper brownish pigmentation in the axillae and gluteal folds. The abdomen is protuberant and tympanitic. There is complete absence of hair in the axillae and pubic regions. The hair of the head is sparse and fine in texture. The mucosa of the mouth, pharynx and conjunctivae is very pale. The sclerae are porcelain white, and the vessels contain no blood. Rigor mortis is complete and there is slight postmortem settling of blood.

Peritoneal Cavity: Subcutaneous fat is reduced to a very small amount. It is a light canary-yellow in color. Abdominal muscles are pale and on section are wet. Lower border of liver lies 6 to 8 cm. below costal margin. Anterior abdominal wall and intestines adjacent to the gallbladder are stained a deep yellow. Pelvis contains a few cubic centimeters of clear, yellowish liquid. Appendix is small, normal in appearance. There is a Treve's fold present. The bladder is distended with urine and reaches to the level of the umbilicus.

Thoracic Cavity: No free liquid, no adhesions; no exudate.

Pericardial Cavity: The pericardium is markedly distended with slightly turbid straw-colored liquid, 120 c.c. in amount.

Heart: The heart is large; right side is dilated; weighs 240 gm. The epicardium is pale and at the base of the heart it encloses clear, yellowish liquid. It is elevated by this liquid to a thickness of 1 to 3 mm., so that the vessels of the surface of the heart appear to be depressed. There is a small amount of bright yellow fat in the epicardium. The myocardium is soft and pale. It is mottled throughout with grayish-yellow areas. The endocardium everywhere presents an almost uniform wavy linear, yellowish marking, running transversely to the direction of the muscle fibers, giving an unusually marked and typical tigroid mottling. The apexes of the papillary muscles are light grayish-yellow in color. The valves and endocardium are normal in appearance. Measurements: tricuspid 9 cm., pulmonary 5.5 cm., mitral 7 cm., aortic 4 cm. The wall of the right ventricle is 7 mm. in thickness; left 12 mm.

Lungs: On removal they partially collapse. Both are pale in color, the anterior halves being pinkish-gray, the posterior halves light salmon-red in color. The lobules are distinctly outlined. The alveoli are prominent and inflated. On the anterior border of both lungs there are areas where the alveoli are markedly distended. On section the cut surfaces yield much thin salmon-red liquid and a small amount of air. The cut surfaces are uniform throughout. The bronchial mucosa is bright pink in color and contains glassy yellow, viscous material.

Gastro-Intestinal Tract: The stomach is normal. Duodenum, jejunum, ileum and colon are normal. Nine *Ascaris lumbricoides* are found in the small intestine. Most of them are immature and range in length from 5 to 10 cm. Other parasites were carefully searched for but were not found.

Liver: This is normal in size; weighs 980 gm.; color is light red with a minute, yellowish mottling. On section the cut surfaces are pale yellowish-red with minute deeper yellowish points and areas which involve the central portion of the lobules. The bile ducts and gallbladder on section are normal.

Pancreas: This organ is normal in size and consistency. On section the cut surfaces have a distinctly yellowish cast. The blood vessels are somewhat prominent as pink dots.

Spleen: This weighs 85 gm.; the capsule is smooth, not wrinkled; the consistency is normal. On section, the cut surfaces are maroon-red in color; the trabeculae are just visible; malpighian bodies appear with normal prominence.

Kidneys: Combined weight 280 gm.; they are large; both are identical in appearance. The perirenal fat has the same yellow color as the rest of the fat in the body. The kidneys are pale in color, normal in shape; the capsule strips readily from its smooth surface. On section the cut surfaces of the cortex appear as pale yellowish and of more than normal opacity. The average width is 6 mm.

The glomeruli are visible as minute colorless dots. The pyramids are pale; otherwise normal. The ureters are normal in size; not dilated.

Adrenals: Both are normal in appearance, except for the presence in the cortex of small nodules 2 to 3 mm. in diameter. Some of these project from the surface of the cortex.

Pelvic Organs: Bladder and genitalia are normal.

Organs of Neck: Tongue, tonsils, pharynx, larynx, trachea and esophagus are normal; thyroid glands are normal; the two parathyroids were dissected out and found to be normal in appearance.

Aorta: Normal throughout. The size is normal for a child of this age.

Head: The subcutaneous tissues of the scalp are markedly thick, due to distention with yellow liquid, which runs freely from the incision. The calvarium, after reflection of the scalp, presents a remarkable appearance. It is dark reddish-purple in color and the surface of the bone is irregular owing to the presence of mound-like elevations or bosses. There are prominent elevations situated symmetrically on the frontal bones and on the parietal bones. These measure roughly 3 cm. in diameter and are elevated approximately 0.5 cm. above the surface of the surrounding calvarium. The bone saws with unusual ease and the surfaces yield much deep-red, pasty marrow. The whole skull is markedly thickened and through the bosses the thickness averages 1 cm. The internal and external tables are very thin, less than 0.5 mm. in thickness, while the thickness of the bone is composed of loose cancelous bone enclosing a large amount of soft, deep-red marrow. The dura and sinuses are normal.

Brain: This is normal in appearance, normal in consistency. Nothing abnormal found on section of the hemispheres, basal ganglia, pons, medulla and cerebellum.

Middle Ears: Normal.

Pituitary: The pituitary body appears normal.

Mesenteric Lymph Nodes: The mesenteric lymph nodes are large, yellowish, with reddish centers. The bronchial lymph nodes are similarly enlarged. The retroperitoneal lymph nodes are enlarged and similar in appearance to the mesenteric. The peripheral lymph nodes are enlarged.

Microscopic Examination.—Heart: The fibers of the inner part of the myocardium show many minute vacuoles; otherwise the myocardium is negative.

Lung: Moderately injected; occasional alveoli contain a few strands of fibrin and numerous polymorphonuclear and large mononuclear cells containing brown granular pigment in about equal numbers. The bronchioles contain masses of polymorphonuclear leukocytes with few mononuclear phagocytic cells.

Spleen: Deeply injected. The malpighian corpuscles are of fair size. The reticular tissue of the pulp seems reduced in amount and contains a relatively small number of lymphocytes. In the sinuses are occasional small groups of cells resembling myelocytes; large mononuclear cells with basophilic cytoplasm. There are also numerous mononuclear and polynuclear eosinophils in the reticular tissue. Occasional nucleated red blood corpuscles are found in the sinuses.

Liver: The centers of the lobules show moderate injection with blood. The liver cells about the central vein covering areas of about a fourth of the radius of the lobule are filled with small vacuoles, giving the cells a spongy appearance. The sinusoids for the most part contain but little blood and no adventitious cells except an occasional mononuclear eosinophil. There is a small amount of light brown pigment in granular form in the liver columns and in the endothelium of the sinusoids.

Kidney: Moderately injected. The glomeruli are negative. The convoluted tubules, distal and proximal, and ascending Henle tubules, show marked dilatation and are filled with circular reticulum and granular material. The epithelium of these tubules is ragged, granular, irregular in amount and faintly stained. Many nuclei are vesicular, small and densely stained. In places the tubules are almost denuded of epithelium, only a narrow fringe of granular cytoplasm remaining.

All types of tubules contain considerable brown granular pigment in the epithelium cells.

Pancreas: Adrenals, thyroid, jejunum, ileum and colon are negative. In the jejunum there is extensive postmortem desquamation of epithelium from the villi.

Bone Marrow of Femur: There is no fat present. The marrow is wholly myeloid tissue with comparatively few normal and nucleated red blood corpuscles. Among the nucleated red corpuscles are many abnormally large ones, of irregular shapes and lobed nuclei (megaloblasts). There are many mononuclear eosinophils (myelocytes) in groups, throughout the sections. The predominately myelocyte occurs in groups and is characterized by a large vesicular nucleus and a small amount of finely granular, basic staining cytoplasm. Mitoses are fairly numerous. Megakaryocytes are sparsely distributed and of small size.

Calvarium: The spaces between the bone trabeculae are filled with active myeloid tissue similar to that found in the femur.

Epicrisis.—The anatomic and microscopic findings in this case admit no other diagnosis than pernicious anemia. The marked thickening of the calvarium, due to a compensatory hyperplasia of the marrow of the diploe, is worthy of note.

The findings of the necropsy justify the clinical diagnosis of pernicious anemia. They show no possible cause for the disease, except the round worms which were found in the intestine. It does not seem reasonable, however, to believe that these worms were the cause of the severe anemia. In the first place, round worms are common in childhood; pernicious anemia is very rare. Moreover, when anemia is apparently due to round worms, it is of a secondary type, and mild. In this instance, while there were a number of worms in the intestines, they were all young. Furthermore, no ova were found in the feces on repeated examinations. It seems reasonable to believe, therefore, that the round worms had been recently acquired and that they had no etiologic connection with the patient's anemia.

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PROGRESS IN PEDIATRICS

RECENT PROGRESS IN OTOLOGIC DISEASES OF CHILDREN *

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During the past twenty-five years progress in the surgical treatment of suppurative otitis media achieved by the classical Schwartz and his followers in Europe, together with many American otologists, has brought this special branch of surgery to the foremost ranks. It has been the means of saving innumerable lives of children. It has saved them from being hopelessly deaf. It has made them selfsustaining and fitted them well for their life work; whereas, prior to this time many of them had been made public charges. While this branch of otology has progressed with great rapidity, not so much can be said for the treatment for the nonsuppurative forms of deafness.

During the past year a great deal of conscientious work has been done along the lines of teaching the deaf child to be a selfsustaining and a useful citizen. Dr. Max A Goldstein[1] of St. Louis, offers a classification of psychological principles in their application in the education of the deaf child, namely: 1. Observation. 2. Concentration. 3. Imitation. 4. Memory. 5. Correlation. 6. Imagination. 7. Rhythm and Mechanics.

1. Observation. First he shows that the faculty of observation in the deaf child is developed largely through the sense of touch or through the sense of sight, and the fact remains that tactile impressions are our most reliable and tangible forces for stimulating the child's mind.

Assuming that the child 5 years of age is totally deaf and has had no special or technical training at this time of his development, he has acquired many of the habits, manners and acts of normal children, but his power of observation has been seriously handicapped by the limited avenues through which his mental functions are stimulated.

Gestures, gymnastics, special movements of the tongue, lips and palate are used for the training of sight. Recognition of form is accomplished by a combination of the sense of sight and touch.

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1. Goldstein, Max A.: The Psychological Study of the Deaf Child, Laryngoscope, 1916, **26,** 1129.

The use of blocks and geometrical solids to stimulate the touch and sight faculties is an effective method of sense training and may be considered in various stages:

(*a*). The child is shown a geometrical solid and is then asked to select same by sight from a number of different shaped solids.

(*b*). The child is directed to feel a solid object while closing the eyes and is then asked to recognize the same object by sight. This correlates his sense of sight and sense of touch.

(*c*). The child is shown a solid object and then selects same by sense of touch with eyes closed. This is the reverse of the second step.

(*d*). From a series of blocks of different shapes the child is asked to select one block by sense of sight. This block is then returned to the rest of the collection and the child directed to select it by sense of touch alone. This sharpens the tactile sense for comparative sizes of lines and surfaces.

(*e*). Two blocks of slight variation in size and shape are presented to the child; he selects one by sense of touch and is then required to find that particular block from a larger group of objects.

In similar manner the pupil learns to differentiate textures and irregularities of surfaces, and the touch sense is so acutely sensitized by constant practice that he is eventually enabled to distinguish the difference in vibrations of musical tones, the differential vibrations of the voice in the throat and chest, which is a vitally important factor in technical training. Not only does this practice develop his powers of observation, but it also stimulates his faculties of concentration, memory and correlation.

2. Concentration. One of the real difficulties encountered in the early training of the deaf child is his lack of concentration. In reading, independent study, and other mental activities, the advanced deaf pupil has the advantage of being able to concentrate his mind on his work without the added extraneous diversions of noise and all forms of sound to which the normal hearing child is subjected. On the other hand, the deaf child not only sees, but also hears through the sense of sight and is constantly on the alert for all visible changes in the schoolroom, and in his surroundings even when attempting to concentrate his attention on the individual work prescribed for him.

When a photographer desires to pose a prattling, restless, lively baby before a camera, he arrests his attention by holding some bright or unusual object before the child at a fixed point. This momentarily arrests the child's attention and gives the photographer his opportunity for a picture. A crying baby is cajoled by a rattle or other sound, a moving, bright object, clapping of the hands, sudden change of position, patting on the body, or any of the other numerous resources

employed to arrest the child's attention. Attention is the basis of concentration; concentration is an indispensable aid to teacher and child.

Our deaf child, therefore, must have his attention engaged in some familiar way by which faculties already alert may be stimulated.

In the classroom a favorite method of developing the power of attention in the young deaf child is the use of matching exercises. Matching colors and objects not only trains the eye, but develops memory and concentration. Even though the child may not know the names of the colors he is asked to compare, he instinctively cultivates a comparative color sense by such practice. By the use of matching charts the teacher studies the dawning of the child's attention faculty and the child becomes more interested in the work of the teacher and in the acquisition, day by day, of new ideas. He cultivates concentration and the pleasure of learning. The more attractive we can make the form and character of the child's daily instruction, the more readily will we get his cooperation.

3. Imitation. One of the fundamental principles in the education of the child is imitation. Imitation is the foundation on which we build gesture, expression, speech and all voluntary movement. It, therefore, is utilized as one of the most valuable aids in child training.

Imitation develops almost simultaneously with observation. The first use of the organs of speech, as the tongue and lips, and the use of the breath, are taught through observation and imitation. The child that has never spoken a word is shown a feather or a slip of paper. The teacher holds the feather or the paper close to the mouth and blows lightly and sharply on it. The child sees the act and feels the impulse of breath and is directed to imitate it. The result is the letter p, t, f or some breath consonant, depending on the position of the lips and tongue. The differentiation of these breath consonants soon becomes apparent and the way is prepared for more complicated sounds.

The positions and movements of the tongue are both observed and imitated. Quick hand movements and gestures give the child additional opportunities for imitation. From the combination of movements of the tongue and lips, speech is gradually evolved. Correct breathing is acquired with somewhat more difficulty. Incidentally, it might be added that insufficient attention has been given to breathing and breath control in the training of the deaf child and in the correction of defects of speech.

Imitation is further accentuated by directing the child to reproduce objects that it sees and movements that are made. Three marbles are held up from a box of marbles; the child is directed to produce the

same number of marbles, and this his faculty of imitation soon enables him to do. Two kindergarten sticks are placed at right angles to each other; the child is asked to reproduce this position, and when this has been effected, he is asked to draw lines on the board in the same position. If he does so correctly from the objects before him, observation and imitation have been called into question. If he draws the object on the blackboard without seeing it, another faculty is being stimulated.

4. Memory. Memory combines the three previous faculties, observation, concentration and imitation. Memory is required to produce speech, to perform an act, to spell a word, to call a name, and to reproduce any process of mental activity.

The normal child first acquires speech by meaningless repetition of some elementary sounds. Before the babe can say "mama" it prattles "mamamama" or sounds like "papapapa" without significance. This principle has been recognized as the basis for teaching speech to the deaf.

Miss Josephine Avondino, of the Central Institute for the Deaf, whose work along this line has been especially intensive, in describing her "system of babbling," says:

> The movements of the muscles of the body of a very young infant are but evidences of the instinctive impulse to act, and, specially, the movements of the lips and tongue and the cooing of a baby are the instinctive preparation for speech. This elementary stage of vocalization soon passes and he reaches the babbling stage. This stage extends over a long period until definite sounds and syllables are gradually acquired.

The cultivation of memory for the acquisition of speech in the deaf child is of even greater importance than memory training for the normal child.

5. Correlation. The essential principles of observation, concentration and imitation are the a, b, c to the acquisition of speech. To this we add memory for the reproduction of speech, but there is still a vital factor necessary to the unfolding and higher development of the child's mentality — the comprehension of speech. A child may be taught the word mother. It may then be told that the word mother applies to a definite person. These two mental impressions may exist independently in the child's mind. The climax of these impressions, however, does not become apparent to the child until he realizes the relationship between the word mother and the person to whom the word applies. This association of ideas, or, as we have classified it, correlation, constitutes one of the most subtle refinements in the psychologic study of the deaf child.

When correlation becomes active in the scholastic work of the deaf child, his mind receives a tremendous stimulus, and from that time we note a decided advancement, not only in his scholastic, but also in his

speech work, facility of expression, and working ambition. A practical example of this unfolding of the child mind is presented in the following case:

E. M., a girl of 7, was admitted to the Central Institute for the Deaf, September, 1914. She was born deaf; functional tests corroborated this statement. There were not even tone islands of hearing; at a distance of 6 inches the child was able to repeat the vowels ah, oh, when called into the ear in a loud voice; the differentiation was uncertain.

This little girl is now in her second scholastic year of oral training; she reads, writes and speaks with fair fluency as compared to a normal child in the same period of training. Her speech is fluent, her voice flexible and well modulated. As soon as this child was able to comprehend, read, and speak phrases and sentences, it occurred to me to apply the principles of reeducation as advanced by Urbantschitsch, Bezold, Gradenigo and others. I was astonished to find after a few trials that this child could perceive spoken words and phrases when called into the ear with a moderate volume of voice. Today she is getting her training not only by speech reading and articulation, but by actual auricular exercises. She can even repeat sentences containing words of which she does not know the meaning and her oral training progresses much more rapidly with the assistance of her increasing hearing capacity.

We have been taught that in the congenitally deaf child the organ of Corti shows but an atrophied remnant of filaments of the cochlear nerve. What, then, has been the reconstructive or regenerative status that has taken place in this child's auditory perceptive mechanism? Reeducation of the deaf implies, as its name would indicate, the restimulation of a sensory apparatus that at some time was active. In this case, however, there had never been an active auditory sense, and the actual hearing capacity of today must be regarded as a new and constructive feature and not as a reeducation or reconstruction. What the psychologic element stimulated in this case might be, I am as yet not prepared to say.

I would say that the marked stimulation in this child's mentality and speech began at the time when the association of ideas or correlation of words and thoughts was first developed.

6. Imagination. Independence and facility of expression are best developed by cultivating the power of imagination. Recently the editor of one or our influential daily newspapers visited the Central Institute and related an interesting story to a number of our pupils of 11 or 12 years of age. The story contained, perhaps, about a thousand words. The raconteur is a man with a full beard and his speech is rather nervous and rapid. Six of our children listened to the story and acquired the subject matter entirely by lip reading. They were given a pad and pencil and asked to write the story as they had received it. The written story by each of the six children was expressed with individuality, conciseness, and accuracy. The essentials were maintained, but in each instance the form and character of the story was

reconstructed in accordance with the mentality and imaginative ability of the pupil. It was interesting to note how much variation could be given by these child minds in this instance. Imagination, as a psychologic element in child training, offers splendid opportunities for independence of expression, and it is in this form of practice that such facility of expression is developed.

M. W., an 8-year-old girl, lost her hearing completely at 3 years of age following an attack of cerebrospinal meningitis. She has been a pupil of the Central Institute for two years. Recently she was given a picture chart and asked to write the story which the picture suggested to her mind.

The imaginative sense expressed by this child is the more remarkable when we consider that her home surroundings are somber and her nature serious. With the stimulation of her imaginative faculty, additional sunshine is injected into her scholastic work and a new impetus given to the child's ambition. Cultivating the imaginative sense materially assists the child in language construction and develops actual, rather than abstract, expression. Imagination stimulates originality and actively assists in enriching the child's vocabulary.

7. Rhythm. An unconscious accessory in child training is the mechanical or automatic correlation of movement in the perception of vocal expression of thought or action. A number of our pupils have been taught to vocalize the musical scale comprising a whole octave. These pupils hear no sound. The notes that they produce with a fair degree of accuracy are evoked by purely mechanical means. They are taught tone placing, the position of the larynx, direction and projection of the voice, the control of breath and the intensity of voice vibration. Their tones are even sustained and mechanically correct. Their control is measured entirely by the tactile sense, especially the sensation of vibration and the differentiation of resonance. Tyndall has made the pertinent observation that light, sound, heat and electricity are all phenomena produced by waves of motion, varying only in degree, quality and intensity.

An ingenious adjunct in the training of the deaf has been introduced by a progressive eastern teacher. By the use of the piano the deaf child is trained to interpret differences in pitch of a vibrating note or chord. We have used this training constantly with our pupils. The pupil stands with his eyes closed and his fingers resting lightly on some part of the wooden piano case, or in some instances the pupils stand with their back to the key-board some distance from the instrument, depending on the transmission of vibrations through the floor. They can differentiate the bass chord from the middle register; a middle register from the upper clef, and these notes or chords can be struck in every variation and interpreted by the pupils with accuracy.

The physical principle involved in this experiment is obvious; the lower the pitch of the note the fewer the number of vibrations per second; the higher the pitch of the note, the more frequent the vibra-

tions per second. It is not the hearing faculty of the pupil, however, which makes this tone differentiation possible, but the actual sharpened tactile sense by which the difference in rapidity of vibration of a sustained tone is perceived.

The value of this mechanical production of the musical scale or the interpretation of the musical vibration to differentiate the pitch of a note has an important and significant application in our work with the deaf. There is a characteristic and almost invariably present monotone in the speech produced by the deaf and it is to overcome this noticeable defect in speech production that we lay unusual stress on the mechanics of voice production. By increased flexibility of voice, greater accuracy in pitch, better volume and more natural placement, we hope to reach such a high degree of efficiency that this characteristic monotone may be eliminated.

Dr. Lester Mead Hubby[2] reports on the "Experimental Treatment of Three Congenitally Deaf Children with Sonorous Vibrations," using an instrument called the Zund-Burguet electrophone, which covers the range of the human voice, and consists of about the same harmonics. By means of the three registers the timbres of three voices, low, medium and high, are produced. The employment of the vibration can be instantly varied to stimulate each ear according to its separate requirements. The intensity of the sound can be ranged sufficiently so that the length of the treatment is not fatiguing to the patient or the operator. The tones can be maintained at a given intensity as long as necessary. In its essential details, the instrument consists of three faradic hammers whose lengths can be altered by means of levers, with telephonic ear receivers.

The author states that in treating these three patients he used in the first case forty-seven treatments, twice on school days and once on Saturdays and Sundays. Each treatment lasted about three minutes. The second patient received fifty-five treatments, at the same intervals, and the third, forty-nine treatments.

He sums up his results as not encouraging so far as sufficient improvement of hearing to be of any practical benefit to the child, yet it would seem that the treatment would be of value in inculcating the concept of tone, and so of inflection, in those just beginning to learn to speak.

Mr. John Dutton Wright,[3] director of the Wright Oral School for the Deaf, New York City, who has done a great deal of work in reeducation of residual hearing, commenting on the Zund-Burguet apparatus, states that the aim of all efforts to increase sound percep-

2. Hubby, Lester Mead: Larngoscope, 1916, **26,** 1152.
3. Editorial: Laryngoscope, 1916, **26,** 1052.

tion is, primarily, to give the patient a greater and easier comprehension of spoken language; that is, to increase the usefulness of the residual hearing. He believes that the problem involves two distinct parts: first, the effort to increase the actual power of perceiving sounds within the range of the speaking voice; and second, the education of the brain more successfully to interpret the meaning of the imperfect sounds perceived.

Mr. Wright, who cooperated with Dr. Hubby in the treatment of the three patients heretofore mentioned, says that all three children were classed in most schools as totally deaf, because there was not a sufficient power of sound perception to differentiate between vowel sounds, no matter how loudly shouted at the closest range, until they had received a long series of exercises for the education of the brain to recognize and discriminate between sounds.

The three patients mentioned had, for some months prior to the series of treatments given by Dr. Hubby, received this educational auricular training, previous to which none of them had been able to recognize any word by ear.

In conclusion, Mr. Wright says that such tests would seem to establish the fact that there are many cases of children too deaf to acquire speech without special instruction and who cannot be benefited by mechanical auricular training, who can, however, be educated to interpret the imperfect sounds that reach them and thus acquire a certain ability to comprehend speech through the ear.

In gaining access to the mind through the ear by means of spoken language we are following the path trodden for millions of years of unnumbered generations. We are, therefore, working in harmony with all inherited and psychologic tendencies and reaching an area of the brain that can be developed in no other way. When this door is wholly closed, we must gain access to the intelligence through the other entrances of sight and touch, but if the main door is even slightly ajar, the sensible thing is to squeeze through, and, if possible, pry it more widely open. Wright says:

> There are many children in this situation in our schools for the deaf, and, owing to the lack of sufficient teachers and perhaps also to inadequate appreciation of the possibilities, they are not getting the educational training for their residual hearing that they should have. The medical profession could do much to change this and to induce those in authority to obtain the necessary assistants and do this valuable educative work.

Dr. Kerr Love[5] says that nearly all middle ear disease of later life, both suppurative and nonsuppurative, commences during the earlier years of the school period. He regards the advent of the aural school

5. Love, Kerr: The Aural School Clinique, Glasgow Med. Jour., February, 1916.

clinic as perhaps the most important development which has taken place in the treatment of ear diseases for the last quarter of a century. The author says that not only is treatment more important in the school period than at any subsequent time, but that it is also very successful.

In Glasgow four well equipped school clinics have been in operation for a period of four years. Practically no operative work is undertaken. Cases requiring removal of tonsils and adenoids are referred to the hospital serving the district in which the child resides. During the four years of its existence about 3,250 cases, or 3 per cent. of the children attending school, have been treated. It was found that in most cases permanent arrest of the discharge was effected without operative treatment of any kind. In others attention to the nasopharynx together with careful treatment of the discharging ears was required. The author says that although the results hitherto obtained were very satisfactory, the best results cannot be secured until the school board assumes the whole responsibility for the treatment of the children's ears, including all the necessary surgical measures.

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CURRENT PEDIATRIC LITERATURE

BACTERIOLOGY AND PATHOLOGY

Bile Ducts, Congenital Obliteration of.
Arch. de méd. d. enfants, 1917, **20,** 363.

Diphtheria Bacilli, Comparison Between Subcutaneous and Intracutaneous Methods of Testing Virulence of.—M. A. Smeeton.
Jour. Infect. Dis., 1917, **21,** 254.

Diphtheria Toxin, Titration of, in Unilaterally Nephrectomized Guinea-Pigs.—H. R. Wahl.
Jour. Infect. Dis., 1917, **21,** 227.

Meningococcus, Cultural Experiments with.—H. W. Crowe.
Lancet, London, 1917, **2,** 201.

Milk, Certified, Presence of Bacillus Abortus Bovinus in.—E. C. Fleischner and K. F. Meyer.
Am. Jour. Dis. Child., 1917, **14,** 157.

Myopathy, Pathologic Histology of Muscles in Severe.—J. C. Navarro and C. A. Correas.
Semana méd., 1917, **24,** 628.

Myositis, Progressive, Multiple, Ossifying.—C. Johannessen.
Norsk Mag. f. Lægevidensk., 1917, **78,** 839.

METABOLISM AND NUTRITION

Feeding, Infant.—M. S. Reuben.
Med. Rec., New York, 1917, **92,** 181.

Feeding, Studies of Infant, Mineral Constituents (Ash) of Milk.—A. W. Bosworth and H. I. Bowditch.

Intestinal Infantilism, Effect of Cod Liver Oil in Growth in Case of.—L. E. Holt, A. M. Courtney and H. L. Fales.
Am. Jour. Dis. Child., 1917, **14,** 222.

Marasmus, Use of Pancreatic Vitamin in Cases of.—W. H. Eddy and J. C. Roper.
Am. Jour. Dis. Child., 1913, **14,** 189.

Milk and Milk Cultures of Bacterium Welchii, Hemolytic Substances in Heated.—W. W. Ford and J. H. Lawrence.
Bull. Johns Hopkins Hosp., 1917, **28,** 245.

Milk, Bacterial Examination of.—F. H. Slack.
Am. Jour. Pub. Health, 1917, **7,** 690.

Milk in Infant Feeding.—E. Fynn.
Semana méd., 1917, **24,** 255.

Milk, Protein, Therapeutic Uses of; Report of Cases.—C. A. Scherer.
Journal-Lancet, 1917, **37,** 574.

Milk, The Necessity for Pure Raw.—H. D. Chapin.
Jour. Am. Med. Assn., 1917, **69,** 886.

Milk, Vacuum Bottles as Receptacles for.—Von Bergen.
Cor.-Bl. f. schweiz. Aerzte, 1917, **47,** 870.

Nutritional Disturbances in Infants.—A. Ravenna.
Riv. clin. pediat., Florence, 1917, **15,** 337.

Scurvy, Importance of Radioscopy in Infantile.—U. Provinciali.
Pediatria, Naples, 1917, **25,** 481.

Scurvy, Twelve Cases of Infantile.—J. Comby.
Arch. de méd. d. enfants, 1917, **20,** 537.

DISEASES OF THE NEW-BORN

Weight, Physiologic Drop in, of the Newly Born.—A. Borrino.
Pediatria, Naples, 1917, **25,** 413.

ACUTE INFECTIOUS DISEASES

B. Diphtheriae, Morphology of Strain of.—P. G. Heinemann.
Jour. Bact., 1917, **2,** 361.

Cerebrospinal Fever, Premeningitic Rash of.—C. P. Symonds.
Lancet, London, 1917, **2,** 86.

Cerebrospinal Meningitis, Epidemic, Histopathology of Internal Ear in Typhoid, Purpura Hemorrhagica and.—F. Tanaka.
Laryngoscope, 1917, **27,** 608.

Diphtheria, Case of Subcutaneous Emphysema in.—Le Soudier.
Arch. de méd. d. enfants, 1917, **20,** 360.

Diphtheria, Present Status of.—R. D. de Sanson.
Brazil-med., 1917, **31,** 185.

Fourth Disease. (Maladie de Dukes.)—A. Lesage.
Presse méd., 1917, **25,** 401.

La Grippe, Discussion of Diagnosis and Treatment of Infantile.—W. W. Dill.
Southwest Med., 1917, **1,** 12.

Measles, One Hundred and Thirty Cases of.—M. A. Moore.
Kentucky Med. Jour., 1917, **15,** 381.

Measles, Purpuric and Cerebral Symptoms Occurring in Defervescence of English; Two Fatal Cases.—T. G. Nicholson.
Lancet, London, 1917, **2,** 87.

Meningitis, Case of Cerebrospinal, Caused by Fränkel's Pneumococcus.—T. Nomura.
Sei-I-Kwai, 1917, **36,** 65.

Meningitis, Epidemic, Mode of Infection, Means of Prevention and Specific Treatment of.—Simon Flexner.
Jour. Am. Med. Assn., 1917, **69,** 817.

Meningitis, Infectious; Study of Twenty-Seven Cases in 586 Necropsies.—S. Graves.
Kentucky Med. Jour., 1917, **15,** 387.

Meningitis, Influenzal.—W. Tobler.
Cor.-Bl. f. schweiz. Aerzte, 1917, **47,** 881.

Meningitis, Mode of Infection, Means of Prevention and Specific Treatment of Epidemic.—S. Flexner.
Jour. Am. Med. Assn., 1917, **69,** 721.

Meningitis, Otogenous, Lumbar Puncture Fluid Constantly Sterile with Severe.—G. V. T. Borries.
Ugesk. f. Læger, 1917, **79,** 997.

Meningitis, Toxic.—Andruetto.
Gazz. d. osp., 1917, **38,** 713.

Poliomyelitis, Acute Anterior.—C. W. Garrison.
Southwest Jour. Med. and Surg., 1917, **25,** 191.

Poliomyelitis, Anterior, Statistical Study; Summary of Recent Work; Orthopedic Treatment.—A. O'Reilly.
Missouri State Med. Assn. Jour., 1917, **14,** 382.

Poliomyelitis, Immune Reactions in Rabbits Injected with Micrococcus from Acute.—G. Mathers and K. Howell.
Jour. Infect. Dis., 1917, **21,** 292.

Poliomyelitis, Massage and Exercise in After-Care of.—F. Berg.
Med. Rec., New York, 1917, **92,** 329.

Poliomyelitis, a Report of a Group of One Hundred and Ten Cases of, from the Acute Onset to the Present Time.—C. Ogilvy.
Jour. Am. Med. Assn., 1917, **69,** 691.

Poliomyelitis, The Orthopedic Treatment of Acute and Convalescent.—E. D. Ebright.
Jour. Am. Med. Assn., 1917, **69,** 694.

Poliomyelitis, Treatment of Anterior, at Fordham Hospital.—S. W. Boorstein.
Jour. Am. Med. Assn., 1917, **69,** 696.

Scarlet Fever, Eye Complications of.—Tito di Giuseppe.
Gazz. d. osp., 1917, **38,** 803.

Scarlet Fever, Surgical Complications of.—F. Bindi.
Gazz. d. osp., 1917, **38,** 755.

Schick Test, with Especial Reference to Negro.—L. T. Wright.
Jour. Infect. Dis., 1917, **21,** 265.

Typhoid in Children, High Calory Feeding in.—H. H. Yerrington.
California State Jour. Med., 1917, **15,** 294.

Whooping Cough, Vaccine Treatment of.—C. J. Bloom.
New Orleans Med. and Surg. Jour., 1917, **70,** 275.

TUBERCULOSIS AND SYPHILIS

Cirrhosis of the Liver, Probably from Inherited Syphilis, in Two Children.—O. Cozzolino.
Pediatria, Naples, 1917, **25,** 385.

Lymphocytosis in Syphilitics.—C. P. Mayer and A. C. Gourdy.
Semana méd., 1917, **24,** 779.

Otitis, Diagnosis of Tuberculous.—M. Lermoyez.
Presse méd., 1917, **25,** 433.

Pretuberculosis in Children and Treatment with Tuberculin Inunctions.—N. R. Blumenau.
Russk. Vrach, 1917, **16,** 289.

Tubercles in the Pons.—A. D'Espine and V. Demole.
Arch. de méd. d. enfants, 1917, **20,** 355.

Tuberculosis and Paroxysmal Thyroid Insufficiency.—E. Albina.
Rev. Asoc. med. argent., 1917, **26,** 967.

Tuberculin, Importance of the Skin Reaction after Inoculation with, as a Guide to Treatment and Prophylaxis.—E. Lanz.
Cor.-Bl. f. schweiz. Aerzte, 1917, **47,** 920.

Tuberculosis in Childhood with Unusual Manifestations.—L. Porter.
California State Jour. Med., 1917, **15,** 303.

GASTRO-INTESTINAL SYSTEM

Appendicitic Abscesses, Retrocecal, in Three Children.—R. A. Rivarola.
Prensa med., Argentina, 1917, **4,** 28.

Appendicitis, Thirty-Three Cases of, in Children.—W. A. Wood.
Med. Jour. of Australia, 1917, **2,** 65.

Diarrhea, Summer, of Babies.—A. T. Coley.
Southwest Jour. Med. and Surg., 1917, **25,** 185.

Digestive Apparatus in Infants, Intoxication with Disease of.—A. B. Marfan.
Nourrisson, Paris, 1917, **5,** 193.

Duodenal Ulcer in Infants; Report of Four Cases.—W. H. Bartran.
Wisconsin Med. Jour., 1917, **16,** 85.

Dyspepsia, Butyric, Mucous Gastritis and Pylorospasm in Young Infants.—F. Chevrel.
Progrès méd., 1917, **32,** 242.

Enterocolitis; Sporadic Dysentery.—R. T. Hocker.
Kentucky Med. Jour., 1917, **15,** 398.

Gastro-Enteritis, Alimentary, and Xerophthalmia in Infants.—S. Monrad.
Ugesk. f. Læger, 1917, **79,** 1177.

Rumination in the First Year of Life.—C. G. Grulee.
Am. Jour. Dis. Child., 1917, **14,** 210.

RESPIRATORY SYSTEM

Bronchus, Foreign Body in.—E. F. Soto.
Rev. med., Havana, 1917, **12,** 353.

Empyema in Childhood.—P. C. Layne.
Kentucky Med. Jour., 1917, **15,** 370.

Respiratory Apparatus, Disease of, in Infants Brought to the Welfare Station. —M. la Ferla.
Pediatria, Naples, 1917, **25,** 469.

Stridor, Chronic, in Infants, Causes of, Four Cases.—M. Lavergne and Donzeau.
Nourrisson, Paris, 1917, **5,** 193.

BLOOD AND CIRCULATORY SYSTEM

Ascites from Liver; Heart Disease in a Child Under Three.—C. Martelli.
Pediatria, Naples, 1917, **25,** 401.

Blood Solids and Concentration of Sodium Chlorid in Plasma, Variation in Infants of.—A. M. Courtney and H. L. Fales.
Am. Jour. Dis. Child., 1917, **14,** 202.

Heart, Graphic and Radioscopic Study of Congenital Disease of.—Five Cases.—E. Lenoble.
Ann. de méd., 1917, **4,** 250.

Leukemia, Acute.—K. Nicolaysen.
Norsk Mag. f. Lægevidensk., 1917, **78,** 769.

Purpura Hemorrhagica, Chronic, and Its Pathogenesis.—F. Schupfer.
Riv. Crit. di Clin. Med., Florence, 1917, **18,** 245.

Venous Murmur.—K. Utheim.
Norsk Mag. f. Lægevidensk., 1917, **78,** 802.

NERVOUS SYSTEM

Babinski Toe Sign.—E. Landau.
Presse méd., 1917, **25,** 424.

Epilepsy, Persistent Treatment of.—H. T. Pershing.
Jour. Am. Med. Assn., 1917, **69,** 869.

Gliosarcoma in an Infant of Seven Weeks, Resembling Hydrocephalus.—L. E. Holt.
Am. Jour. Dis. Child., 1917, **14,** 219.

GENITO-URINARY SYSTEM

Hematuria with Kidney Stones.—Four Cases.—A. P. Martin.
Siglo méd., 1917, **44,** 568.

Phenolsulphonephthalein Elimination in Infants and Young Children.—J. C. Gittings and A. G. Mitchell.
Am. Jour. Dis. Child., 1917, **14,** 174.

Pyelitis, Experimental in the Rabbit.—H. F. Helmholz and C. Beeler.
Jour. Am. Med. Assn., 1917, **69,** 898.

Vulvovaginitis.—A. L. Beckett.
Kentucky Med. Jour., 1917, **15,** 367.

OSSEOUS SYSTEM

Bones, Localization of Growing Point in Epiphyseal Cartilage Plate of.
Am. Jour. Orthop. Surg., 1917, **15,** 563.

SKIN AND APPENDAGES

Burns and Scalds as a Cause of Child Disablement and Death.
Dublin Jour. Med. Sc., 1917, **143,** 313.

Dermographism in Diagnosis.—L. Schwartz.
Cor.-Bl. f. schweiz. Aerzte, 1917, **47,** 817.

Ichthyosis Family, An.—V. Magnus.
Norsk Mag. f. Laegevidensk., 1917, **78,** 703.

Impetigo Contagiosa, Treatment of.—H. Morrow.
Jour. Am. Med. Assn., 1917, **69,** 176.
Perleche.—J. E. Lane.
Jour. Am. Med. Assn., 1917, **69,** 192.
Psoriasis, Bullous.—A. Kissmeyer.
Hospitalstidende, 1917, **60,** 717.
Scabies, Diagnosis of.—R. Sabouraud.
Presse méd., 1917, **25,** 364.
Skin Defects, Congenital.—I. A. Abt.
Am. Jour. Dis. Child., 1917, **14,** 113.
Urticaria.—C. T. Drennan.
Arkansas Med. Soc. Jour., 1917, **14,** 57.
Urticaria, Experiments on Local. Endermic Reactions.—T. Sollmann.
Jour. Pharm. and Exper. Therap., 1917, **10,** 147.

EYE, EAR, NOSE AND THROAT

Blind, What State of Illinois Is Doing for Its.—C. E. Comstock.
Modern Hosp., 1917, **9,** 80.
Conjunctivitis, Vernal, Some Remarks Concerning.—F. Allport.
Ophth. Rec., 1917, **26,** 395.
Ear, Factors Involved in Excavation of Cavities in Cartilaginous Capsule of, in Human Embryo.—G. L. Streeter.
Am. Jour. Anat., 1917, **22,** 1.
Intubation. Fastening the Tube in the Throat.—E. Bernasconi.
Rev. méd. de la Suisse romande, 1917, **37,** 144.
Lypodystrophia Progressiva.—F. P. Weber.
Brit. Jour. Child. Dis., 1917, **14,** 81.
Middle Ear, Acute Inflammation of.—J. H. James.
Journal-Lancet, 1917, **37,** 474.
Optochin Amblyopia.—I. Schiøtz.
Norsk Mag. f. Laegevidensk., 1917, **78,** 717.
Otitis Media, Acute, Indications for Operation in.—E. B. Dench.
Jour. Am. Med. Assn., 1917, **69,** 878.
Otitis Media, Acute, of Diphtheritic Origin.—W. C. Bane.
Laryngoscope, 1917, **27,** 626.
Otitis Media, Acute, with Paralysis of Sixth Nerve (Gradenigo Symptom Complex).—D. Guthrie.
Edinburgh Med. Jour., 1917, **19,** 15.
Otitis Media, Dry Hypochlorite and Boric Acid Treatment of Chronic.—A. Pugnat.
Bull. de l'Acad. de méd., Paris, 1917, **78,** 19.
Retinochoroiditis, Juxtacapillary.—M. I. Puiggari.
Prensa med., Argentina, 1917, **4,** 30.
Sclerotics, Blue, in Connection with Other Anomalies.—N. Voorhoeve.
Nederl. Tijdschr. v. Geneesk., 1917, **1,** 1873.
Sinusitis in Infants, Diagnosis and Treatment of.—R. H. Skillern.
Jour. Am. Med. Assn., 1917, **69,** 895.
Throat, Extraction of Brooch in, of Seven Months Infant for Thirty-Four Hours.—E. Apert.
Nourrisson, Paris, 1917, **5,** 230.
Tonsil, Lingual, Hypertrophy of.—G. Turtur.
Policlinico, Rome, 1917, **24,** 813.
Tonsillectomy, The Foundations of Voice Impairment Resulting from.—E. L. Kenyon.
Jour. Am. Med. Assn., 1917, **69,** 709.
Tonsils as Focus of Infection.—J. M. Brown.
California State Jour. Med., 1917, **15,** 290.
Trachoma—Diagnostic Point and Method of Treatment.—V. E. VanKirk.
Pennsylvania Med. Jour., 1917, **20,** 708.